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THE PSYCHOLOGICAL REVIEW

THE DETERMINERS OF BEHAVIOR AT A CHOICE POINT¹

BY EDWARD CHACE TOLMAN

University of California

The question I am going to discuss is the very straightforward and specific one of 'why rats turn the way they do, at a given choice-point in a given maze at a given stage of learning.'

The first item in the answer is fairly obvious. They turn the way they do because they have on the preceding trials met this same choice-point together with such and such

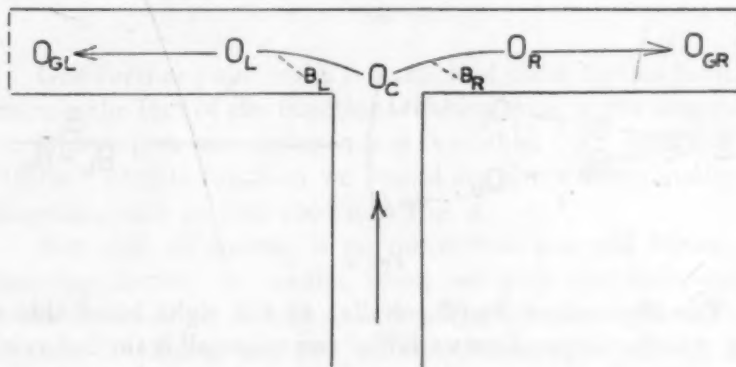


FIG. 1

further objects or situations, down the one path and down the other, for such and such a number of preceding trials. Let me, however, analyze this further, with the aid of a couple of diagrams. First, consider a diagram of a single choice point (Fig. 1).

¹ Presidential address delivered before the American Psychological Association, Minneapolis, September 3, 1937.

In this figure the point of choice itself is designated as O_c ; the complex of stimulus-objects met going down the left alley, as O_L , that met going down the right alley, as O_R ; the goal at the left, as O_{GL} ; and that at the right, as O_{GR} . The behavior of turning to the left is represented by the arrow B_L ; and that of turning to the right, by the arrow B_R . And the point I am now making is that the relative strength of the tendency to turn, say, left (rather than right) will be, first of all, a result not only of the present presentation of O_c but also of all the previous presentations of it together with the O_L , O_{GL} , O_R , and O_{GR} consequences of having behaved by B_L and B_R on all these preceding occasions. In short, I would schematize this feature of the causal determination of the left-turning tendency by the diagram shown in Fig. 2.

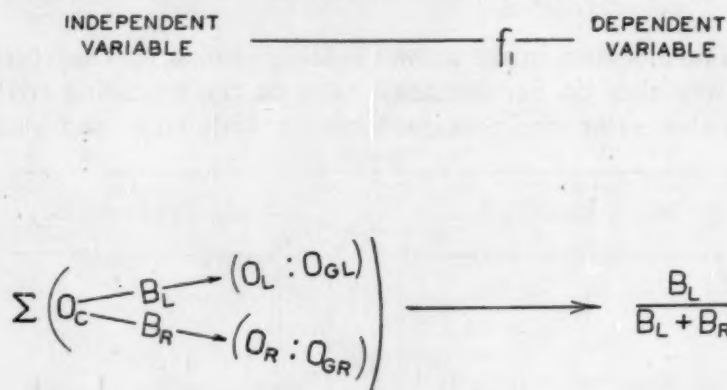


FIG. 2

The expression $B_L/(B_L + B_R)$ at the right hand side of Fig. 2 is the 'dependent variable' (we may call it the behavior-ratio). It is the percentage tendency at any given stage of learning for the group as a whole to turn left. And the hieroglyphic at the left hand side of this figure is the 'independent variable' which determines this behavior-ratio. This hieroglyphic is to be read as meaning: the *sum* of all the preceding occasions in which O_c has, by virtue of B_L , been followed by O_L and O_{GL} and by virtue of B_R been followed by O_R and O_{GR} . This diagram is thus no more than a schematic

way of representing the, shall we say, (to use the term we theoretical psychologists have of late taken so violently to our bosoms) 'operational' facts. The expression at the left is an 'operationally defined' independent variable and that at the right, an 'operationally defined' dependent variable.

For brevity's sake, I shall often substitute, however, an abbreviated symbol for the left-hand term, viz.: simply $\Sigma(OBO)$, as shown in Fig. 3.

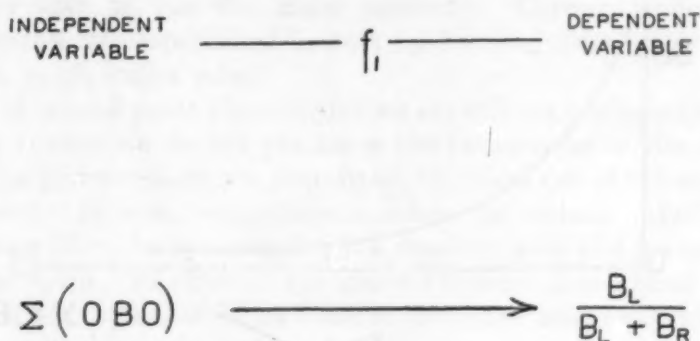


FIG. 3

One further point—the f_1 in each of these figures indicates merely the fact of the functional dependence of the dependent variable upon the independent variable. To indicate the "form" of this function we would require a more analytical diagram, such as that shown in Fig. 4.

But this, of course, is no more than our old friend, the learning curve. It results when we plot the independent variable along an X axis and the dependent variable along a Y axis. Nothing very new so far. It seems surprising, however, that in spite of the thousands, not to say millions, of such learning curves which have been obtained in the last four decades in American rat laboratories there are still a variety of quite simple things about this function which we do not yet know or with regard to which we are still in dispute.

For example, we are still in dispute, first of all, as to the relative importance of the occurrences of the two alternative behaviors B_L and B_R , where B_L is 'wrong' and B_R is 'correct.' (See Fig. 5.)

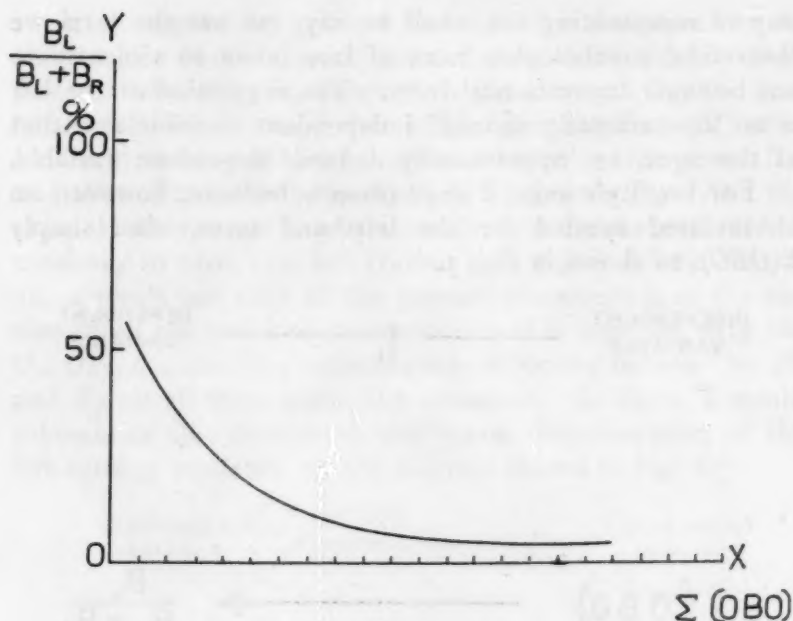


FIG. 4

Thorndike (118, 119) and Lorge (69) and their co-workers, as you all know, working with human beings in analogous, though verbal, set-ups have now concluded that the occur-

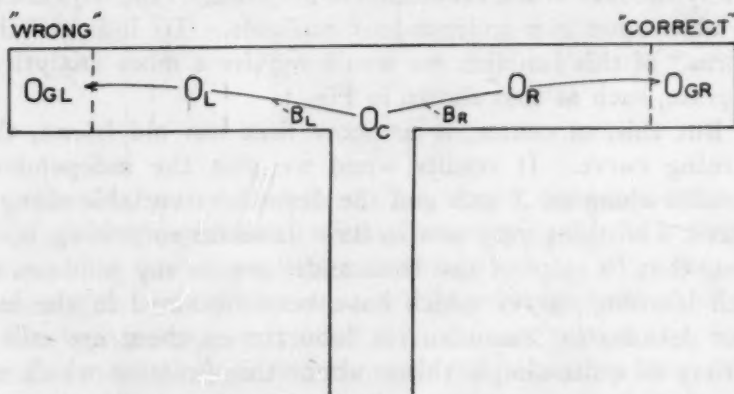


FIG. 5

rence of the wrong behavior has no such general causative effect. They find that learning appears only as a result of the occurrences of the rewarded sequence $O_C - B_R \rightarrow (O_R : O_{GR})$.

On the other hand, still more recently, Muenzinger and Dove (95), working with set-ups similar to Thorndike's have found that the occurrence of the wrong response $O_C - B_L \rightarrow (O_L : O_{GL})$ does weaken its tendency to re-occur. Also Carr, as a result of a series of experiments done by his students (54, 72, 132, 135, 137) some time since in the Chicago laboratory, was finally forced to conclude that "... a certain number of errors must be made and eliminated before the subject is ever able to run the maze correctly. Correct modes of response are established in part by learning *what not to do*" (16, p. 98, italics mine).

A second point about which we are still surprisingly ignorant is that we do not yet know the importance of the rat's being permitted, or not permitted, to return out of the wrong choice. In some experiments, when the animal takes the wrong alley, he passes through a one-way gate and is started over again. In others, he is allowed to treat it as a blind and back out. But, so far as I know, there has been no carefully controlled comparison between these two procedures.

Thirdly, the question of the relative effects of concentrated versus distributed repetitions has not as yet received the thoroughgoing experimental analysis that it deserves. But I understand that Professor Stone and his coworkers are now directing their attention to it and are getting some very significant findings.

Fourthly, we are ignorant concerning the difference between animals which have an initial left-hand bias and those which have an initial right-hand bias.² We usually lump the results for both types together in a single curve. But we might well separate them and study them independently.

Fifthly, Brunswik (14) has recently brought to light a new point in our ignorance. He has been trying the effect of rewarding on the right and rewarding on the left different proportions of times. In other words, it was no God-given rule but apparently some merely human predilection on our

² For one of the first experiments indicating that there are such biases, see Yoshioka (149).

part which made us heretofore tend almost invariably to make one of the alternative behaviors always rewarded and the other always punished. But other frequencies of reward and punishment are equally possible and equally deserving of study.

Sixthly, experiments by Krechevsky (59, 60, 61), seem to indicate that there may be certain general features about the content of the *OBO*'s such, for example, as their containing variable or non-variable paths, which are very important in determining the resultant behavior-ratios and about which we need more information.

Seventhly, a further point which needs more investigation is, as Muenzinger and his coworkers (87, 88, 89, 90, 91, 92, 93) have beautifully brought out, the fact that punishment or obstacles to be overcome, *even on the correct side*, may sometimes seem to aid rather than hinder learning. (See also Tolman, 125, and Tolman, Hall and Bretnall, 127.)

Eighthly, there is the question of what happens when $\Sigma(OBO)$, the number of trials, has become very great. This seems to induce a special sort of result for which the term 'fixation' has been suggested.³ And further studies of such 'fixations' are needed.

Ninthly, the problem as to the effect of temporal intervals between O_C and the resultant O_{GL} and O_{GR} are still by no means altogether completely worked out in spite of all the beautiful work of Hunter and his students, and others who have followed after, on the 'delayed reaction' and on 'double alternation.'⁴

Finally, however, there is a point with regard to which we are not altogether ignorant but the importance of which we usually overlook—namely, the fact that any such function—any such learning curve, actually, is always obtained within the matrix of a larger number of other independent variables

³ See the original experiments on fixation by Gilhousen (31, 32), Krechevsky and Honzik (62) and Hamilton and Ellis (28, 38, 39).

⁴ The literature on these matters is, of course, already enormous and I can not pretend to quote it here. It will suffice to refer to Munn's chapter on 'Symbolic Processes' (95, Chap. VII) and to Heron's chapter on 'Complex Learning Processes' (40).

in addition to $\Sigma(OBO)$. The following is a tentative list of such other variables together with $\Sigma(OBO)$:

I. ENVIRONMENTAL VARIABLES

M —Maintenance Schedule

G —Appropriateness of Goal Object

S —Types and Modes of Stimuli Provided

R —Types of Motor Response Required

$\Sigma(OBO)$ —Cumulative Nature and Number of Trials

P —Pattern of Succeeding and Succeeding Maze Units

II. INDIVIDUAL DIFFERENCE VARIABLES

H —Heredity

A —Age

T —Previous Training

E —Special Endocrine, Drug or Vitamin Conditions

As you will see, I have divided such independent variables into two groups which I have called: (I) Environmental Variables, and (II) Individual Difference Variables. The *environmental variables* are M , the maintenance schedule, by which I mean time since food, water, sex, parturition, or the like, which in common parlance we would call the drive condition; G , the appropriateness of the goal-object provided at the end of the maze relative to this drive; S , the specific types and modes of stimuli which the maze provides; R , the specific kinds of motor response required of the animal in the maze; $\Sigma(OBO)$, the cumulative sum and manner of trials; and P , the general pattern of the maze, that is to say, the number and sorts of preceding and succeeding units. The individual difference variables are: H —heredity, A —age; T —previous training, and E —any special endocrine, drug, or vitamin conditions.

But if, now, we are to include all these independent variables together with $\Sigma(OBO)$, we must have a new causal picture. I suggest the one shown in Fig. 6.

A main causal line has been drawn, as you see, issuing from each environmental variable. And the individual dif-

ference variables, *H*, *A*, *T*, and *E*, have been arranged as possible modifiers of each such main causal line. And what I have hereby tried to indicate is merely the actual types of experiment which we maze-psychologists go in for.

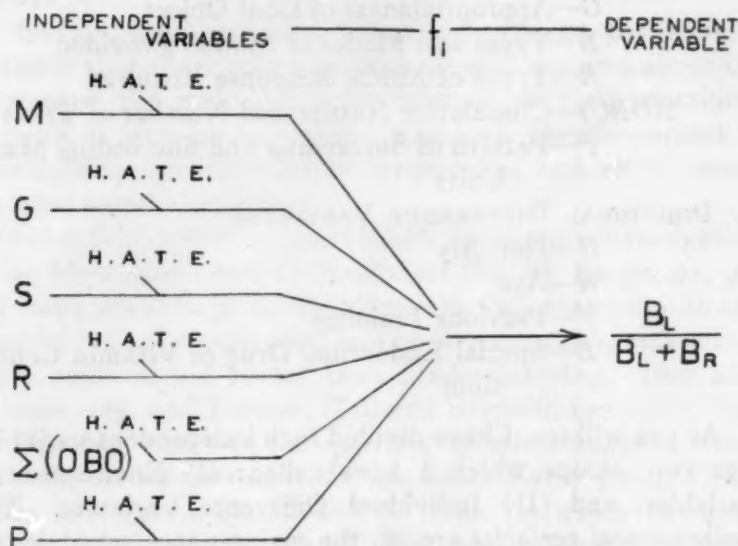


FIG. 6

I wish now, however, to pass from the above outline of experiments to a consideration of theories. But why, you may ask, can we not be satisfied with just experiments and the 'facts' resulting from them?⁵ I find that there are two reasons. In the first place, an entirely factual, empirical establishment of the complete functional relation, f_1 , to cover the effects on $B_L/(B_L + B_R)$ of all the permutations and combinations of *M*, *G*, *S*, etc., etc., would be a humanly endless task. We have time in this brief mortal span to test only a relatively limited number of such permutations and combinations. So, in the first place, we are forced to propose theories in order to use such theories to extrapolate for all these combinations for which we have not time to test.

⁵That the facts must be obtained first of all and that we psychologists have for the most part been both extremely lazy and extremely shoddy in our pursuit of the 'facts' has been eloquently pointed out by Brown (12).

But I suspect that there is also another reason for theories. Some of us, psychologically, just demand theories. Even if we had all the million and one concrete facts, we would still want theories to, as we would say, 'explain' those facts. Theories just seem to be necessary to some of us to relieve our inner tensions.

But what is a theory? According to Professor Hull (49), a theory is a set of definitions and postulates proposed by the theorist (on the basis presumably of some already found facts) from which other empirically testable facts, or as he calls them, theorems, can be logically deduced. These deduced theorems will be new empirical relationships which the theorist—or more often, his research assistants—can, then and there, be set to look for.

For my own nefarious purposes, however, I wish to phrase this matter of the relationship of a theory to the empirical facts out of which it arises and to which it leads in somewhat other terms. A theory, as I shall conceive it, is a set of 'intervening variables.' These to-be-inserted intervening variables are 'constructs' which we, the theorists, evolve as a useful way of breaking down into more manageable form the original complete f_1 function. In short, I would schematize the nature of our psychological theories by Fig. 7. In place of the original f_1 function, I have introduced a set of intervening variables, I_a, I_b, I_c , etc., few or many, according to the particular theory. And I have conceived a set of f_2 functions to connect these intervening variables severally to the independent variables, on the one hand, and an f_3 function to combine them together and connect them to the final dependent variable, on the other.⁶

But turn, now, to some of the actual theories. I shall restrict myself to the discussion of three—Professor Thorndike's, Professor Hull's, and my own. This, of course, will hardly be a fair survey of the field. There are many other doctrines of learning, as, for example, Professor Guthrie's (33),

⁶ For previous presentations of this notion of 'intervening variables' see Tolman (124, 126).

and those of the other conditioned reflex psychologists (145)⁷ and those of the Gestalt school, (2), (45), (55), (56), (143), which are of as great importance and which have equally

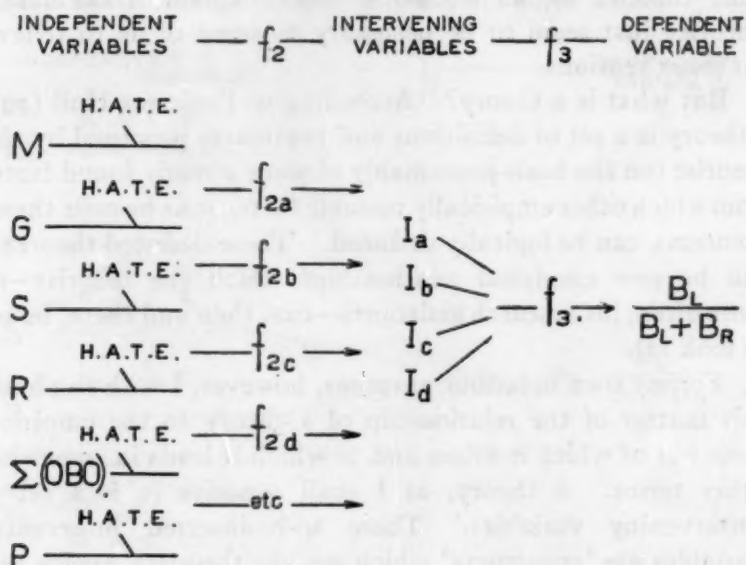


FIG. 7

affected my own thinking. But I shall have to omit a discussion of them here.

Professor Thorndike's 'intervening variables' are quite simple. They are 'stimuli,' 'bonds' or 'connections,' and 'response-tendencies.' His theory I would represent, therefore, by the diagram shown in Fig. 8. It is Thorndike's conception of the nature of the f_2 function which seems to be the crux of his theory. Originally, his statement of this function included both a Law of Exercise and a Law of Effect. But now, as we all know, it includes a Law of Effect only, and a truncated law at that. For, as now stated, Thorndike finds that it is the repetitions of the rewarded sequence $O_C - B_R \rightarrow (O_R : O_{GR})$ which alone are important. These strengthen the C_R connection. The repetitions of the

⁷ For a superb presentation and summary of all the conditioned reflex theories of learning see Hilgard (44).

punished $O_c - B_L \rightarrow (O_L : O_{GL})$ sequence do not, he says, correspondingly weaken the C_L connection.

I have quite a number of quarrels with this theory. I would like to say first, however, that it seems to me that this

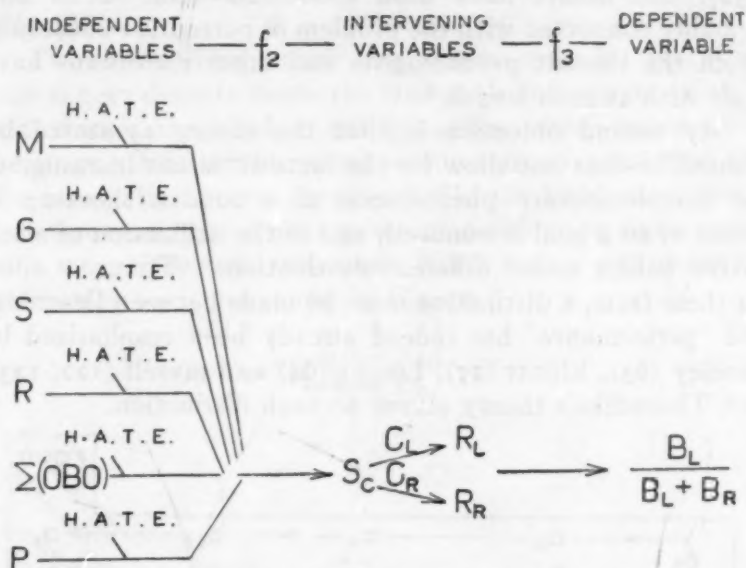


FIG. 8

theory of Thorndike's either in its present or in its earlier form, is *the* theory relative to which the rest of us here in America have oriented ourselves. The psychology of animal learning—not to mention that of child learning—has been and still is primarily a matter of agreeing or disagreeing with Thorndike, or trying in minor ways to improve upon him. Gestalt psychologists, conditioned-reflex psychologists, sign-gestalt psychologists—all of us here in America seem to have taken Thorndike, overtly or covertly, as our starting point. And we have felt very smart and pleased with ourselves if we could show that we have, even in some very minor way, developed new little wrinkles of our own.

Let me now, nonetheless, try to present my criticisms. First, Thorndike's theory, as I see it, identifies stimuli (S 's) with gross objects (O 's) and identifies specific muscular

responses (R 's) with gross means-end behaviors (B 's). And this procedure seems to me to require more justification than he gives it. It raises the problem of 'equivalence of stimuli' and 'equivalence of response' which Klüver (53), Waters (138), and others have been concerned with. It is also probably connected with the problem of perception-constancy which the Gestalt psychologists and other Europeans have dealt with at such length.*

My second objection is that the theory as stated by Thorndike does not allow for the facts of 'latent learning,' of the complementary phenomenon of a sudden shoot-up in errors when a goal is removed, and of the utilization of alternative habits under different motivations. That, to allow for these facts, a distinction must be made between 'learning' and 'performance' has indeed already been emphasized by Lashley (63), Elliott (27), Leeper (64) and myself (122, 123). But Thorndike's theory allows no such distinction.

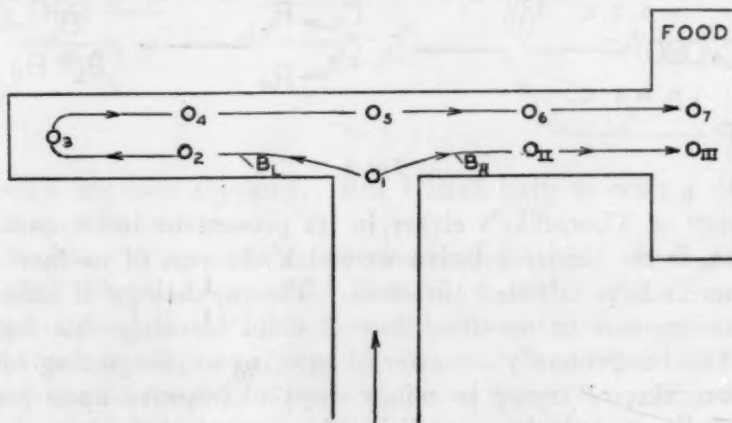


FIG. 9

Finally, my third objection is that the theory does not, for the most part, make anything of the other circumambient variables M , G , S , etc., in addition to $\Sigma(OBO)$. No doubt Thorndike, if this were pointed out to him, would try to work all these other independent variables in as further conditions tending to favor or hinder the respective strengths of C_R and C_L . But my suspicion is that he would have difficulty.

* For a resume of this work see Koffka (56, Chap. VI).

Turn, now, to Professor Hull's theory. For Hull the intervening variables are 'conditionings' of the running responses to successive aggregates of exteroceptive, proprioceptive, and interoceptive stimuli. In order to explain this, first let me present another picture of the simple *T*-maze (Fig. 9).

Two alternative routes are shown—one in which the animal goes directly down the true path and one in which he first chooses the blind to the left. Successive points along these two paths are indicated as successively numbered *O*'s. The true path involves three such *O*'s, the blind alley, seven, and, to explain the tendency which develops in such a situation to go right rather than left, Hull's theory postulates the intervening variables shown in Fig. 10.

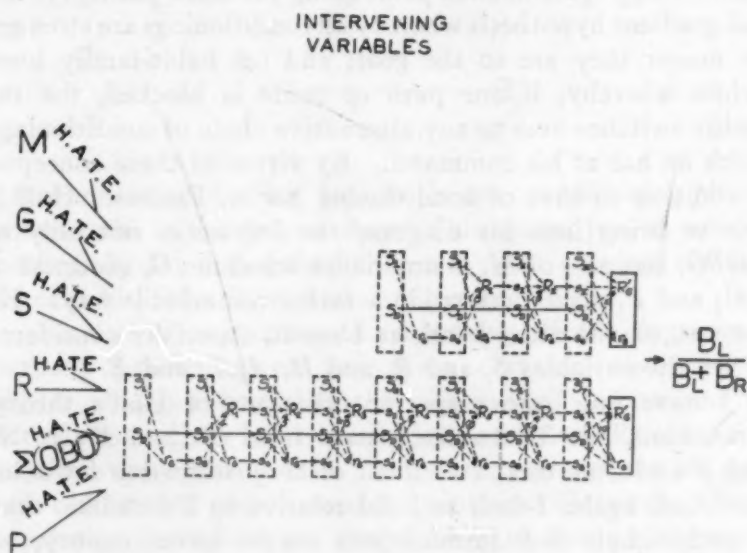


FIG. 10

What I have done, as you will see, is to insert one of Hull's own diagrams (48, 44) in the middle and to call it his set of intervening variables. You are all familiar with such diagrams. They are very clever and can be invented, as I know to my cost, to explain practically any type of behavior,

however far distant from an instance of conditioning such a behavior might at first sight appear. I have, therefore, the greatest respect for them. And, even though I argue against them, I find myself continually being intrigued and almost ready to change my mind and accept them and Hull after all.

It must be noted further, however, that there are certain other concepts besides conditioning involved in these diagrams which help to make them work. These seem to be: (1) anticipatory goal-responses, *i.e.*, the little r_g 's with their little resultant proprioceptive or interoceptive s_g 's whereby the character of the goal is brought back into the aggregates of conditioned stimuli at the different points along the maze; (2) the continuous drive stimulus S_D which also appears at all points and thus also becomes part of the total conditioned stimulus-aggregate at each point along the maze paths; (3) the goal-gradient hypothesis whereby all conditionings are stronger the nearer they are to the goal; and (4) habit-family hierarchies whereby, if one path or route is blocked, the rat readily switches over to any alternative chain of conditionings which he has at his command. By virtue of these concepts, in addition to that of conditioning *per se*, Professor Hull is able to bring into his diagram the influences not only of $\Sigma(OBO)$ but also of M , maintenance schedule; G , goodness of goal; and P , maze-pattern, in a rather remarkable way. He has not, on the other hand, as I see it, especially considered as yet the variables S , and R , and H , A , T , and E .

I have four rather specific criticisms of Hull's theory. First, Hull, like Thorndike, passes from O 's and B 's to S 's and R 's with no clear statement of his justification for doing so. And, again, I feel, as I did relative to Thorndike, that, if such simple S - R formulations are to have cogency, we must be told why and how the actual gross O 's can be reduced to simple S 's, and the actual gross means-end B 's to simple R 's.

My second criticism lies in the fact that I doubt that the supposed laws of conditioning are as simple and as well-known as Hull assumes. Many of the actual workers in the field, for example Loucks (70, 71), Liddell (67), Culler (21),

Schlosberg (105, 106), Hilgard (42, 43) seem to find conditioning a very variable and complicated phenomenon. To explain maze behavior by conditioning seems to me, therefore, like asking the halt to lead the blind. Or to put this another way, what Skinner (108) (see Fig. 11) calls his Type I sort

TYPE I



TYPE II

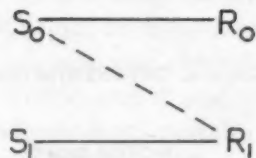


FIG. 11

of conditioning (which for me is not conditioning at all) seems to be at the present stage of the game, just as well and perhaps better understood than the more classical, or what he calls his Type II, sort of conditioning.

Finally, when it comes to using one of Hull's diagrams for actually predicting, on any given occasion, the value of $B_L/(B_L + B_R)$ I find that the difficulty of determining the actual strengths to be assigned to the various S-R connections an almost insuperable one. But, then, perhaps an analogous sort of criticism will be raised against my diagrams. So, in conclusion, let me repeat that I have a tremendous respect for Professor Hull's theory and that I am not by any means as yet altogether certain that mine is better.

I come, now finally, to my own theory. But first, I would like to make it clear that however complicated what I am actually going to present may appear, it will be in reality an *over-simplified and incomplete* version. Partly for the sake of simplicity and partly also, I suppose, because I have not as

yet completely thought the whole thing through, the diagrams I shall present will not contain as many 'intervening variables' nor as complicated interfunctional relations as, I suspect, will finally actually prove necessary. They will, however, indicate the general picture.

My first diagram would be that shown in Fig. 12.

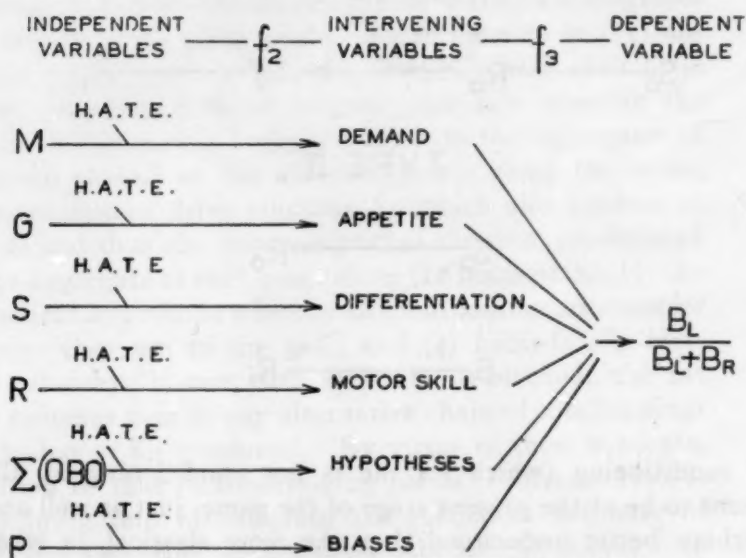


FIG. 12

Note the list of *intervening variables*: 'demand,' 'appetite,' 'differentiation,' 'skill,' 'hypotheses,' and 'biases.'⁹ Such concepts are, I am sure, irritating in that they appear subjective and not the sort to be permitted in an honest behaviorism. Each of them is, nonetheless, I would claim, capable of a perfectly objective definition and measurement. Thus, you will note that each is depicted as resulting from its own correlative environmental variable plus the controlling

⁹In addition to these the final version of the theory would, I suspect, have to add other intervening variables such as: 'general activity,' for the best discussion of this which I know see Munn (97, Chap. II); general attentivity or 'vigilance,' see Krechevsky (58); and demand for 'parsimony'—i.e., demand against 'distance' and 'barriers,' see, for example, Tolman (122, Chap. VII), Gengerelli (30), McCulloch (64), Tsai (133), Waters (140), Wheeler (143) and Wright (148).

effects of H , A , T , and E . 'Demands' result from M 's; 'appetites' from G 's; 'differentiations' from S 's; 'skills' from R 's; 'hypotheses' from $\Sigma(OBO)$'s; and 'biases' from P 's. And I am now going to assert that each such 'intervening variable' is defined by a standard experiment in which its correlative independent environmental variable is systematically varied. Further, in each such experiment all the other independent variables are held constant while the one in question is systematically changed. Under such conditions the resultant variations in $B_L/(B_L + B_R)$ are, by definition, to be said to mirror directly the variations in the one given intervening variable.

For example, the intervening variable—'demand'—(say for food) shall, by definition, be measured by the variations in the behavior-ratio which occur in a standard experiment when G and S and R and $\Sigma(OBO)$ and P and H , and A , and T , and E , that is, all the independent variables *other than* M , are held constant at certain 'standard' values, while M , itself, is systematically varied. For example, as standard values for these other variables I should probably choose: for G the regular standard living diet of the colony, for S an elevated

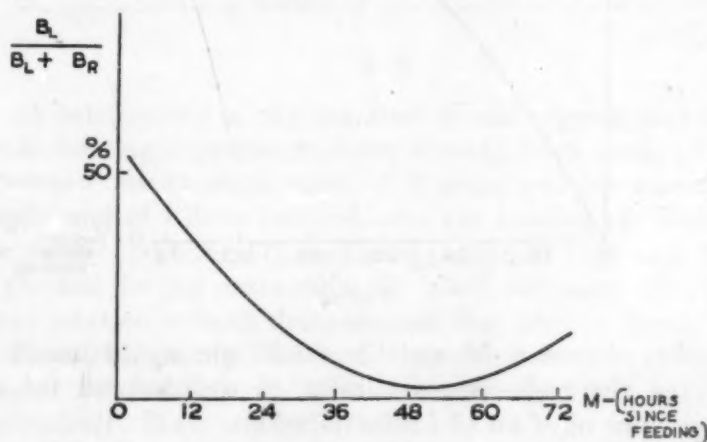


FIG. 13

maze in which all possible visual, olfactory, auditory, tactual and kinæsthetic stimuli would be available, for R a maze

which involved running rather than swimming, or climbing, or going hand over hand, or pulling strings, or what not, for $\Sigma(OBO)$, that set-up which makes the left-hand side a blind and a distribution of one trial every 24 hours, and a number of trials which, for an average value of M , would bring the learning curve about down to the base line—say some 10 trials—and for P a single-unit T with no preceding or succeeding units. With such a set-up in which all the other independent variables would thus be given these standard values and held constant, I would then vary M and study the correlated variations in $B_L/(B_L + B_R)$. And the sort of results one would get are shown in Fig. 13.

But the demand should really be defined as *inversely* related to this $B_L/(B_L + B_R)$ ratio, so that replotting one would have as one's final defining function that shown in Fig. 14. And having, thus at last, this curve—this f_2

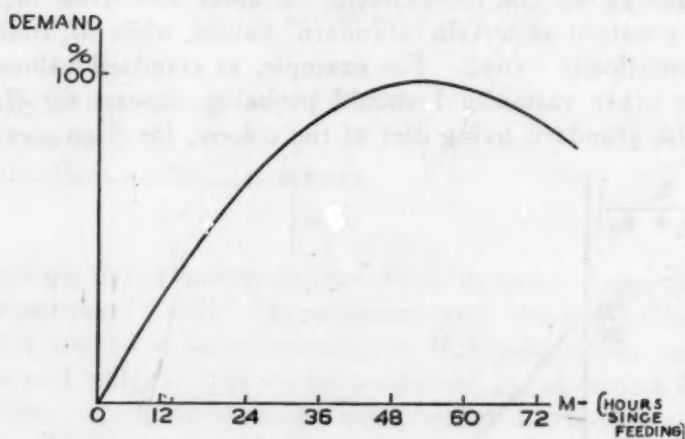


FIG. 14

function—between M and 'demand' one would use it for defining the to-be-assumed value of the demand for any given value of M on all future occasions.

But this procedure, which I have thus outlined in some detail for demand, could also be used in analogous fashion for defining each of the other intervening variables. For each of them, also, we could set up a defining experiment in which

all the independent variables other than the correlative one, would be held constant while that one was systematically varied. And we would obtain in each case a resultant defining curve or table. Figure 15 schematizes the fact of such possible defining procedures.

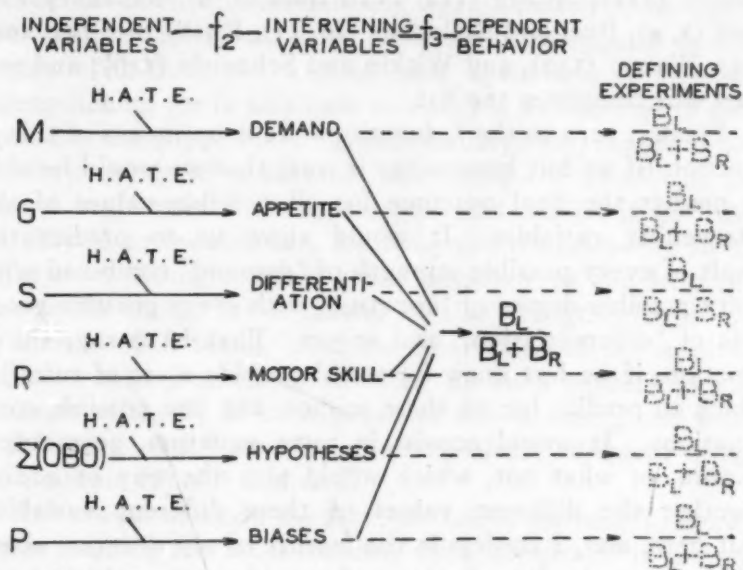


FIG. 15

A brief review of the literature would suggest that many such defining experiments have already been done. Under 'demand' we think at once of Warden and his co-workers (136), and of Elliott (26). Under the heading of 'appetite' we think of Young (151, 152, 153), Elliott (25) and Bruce (13), and for an extraordinarily good summary of all the work relative to both demands and appetites we would look to Stone's chapter in Moss's 'Comparative Psychology' (115). Under the heading of 'differentiation' we think of many individuals: Watson (141), Carr (17, 18, 19), Hunter (50, 51), Dennis (24), Casper (20), Lindley (68), Wolfle (147), and Honzik (46), to mention only a few. Under the heading of 'motor skill' we think of Macfarlane (73). Under that of 'hypotheses' we think of practically all rat-runners in the

world but for the final indignity of suggesting such a term as 'hypotheses' we must blame Krechevsky (57).¹⁰ And finally, under 'biases' we think of Dashiell (22), Bayroff (6), Dashiell and Bayroff (23), Schneirla (107), Yoshioka (149, 150), Ballachey and Krechevsky (5), Spence (110), Spence and Shipley (111), Spragg (112, 113), Buel (7, 8), Ballachey and Buel (3, 4), Buel and Ballachey (10, 11), Ruch (100, 101, 102, 103), Waters (139), and Witkin and Schneirla (146); and not even this completes the list.

Finally, turn to the f_s function. It is by means of this f_s function (if we but knew what it was) that we would be able to predict the final outcome for all possible values of the intervening variables. It would allow us to predict the result of every possible strength of 'demand' combined with every possible degree of 'appetite,' with every possible goodness of 'differentiation,' and so on. That is to say, the f_s function, if we but knew it, would provide a set of rules by which to predict for all these million and one possible combinations. It would consist in some equation, geometrical picture, or what not, which would give the way of adding together the different values of these different variables. But here, alas, I confess is the feature of my doctrine about which I am, to date, haziest. I would venture, however, a few suggestions.

First I would assert that the implicit assumption of most other psychologists is to the effect that their f_s functions are in the nature of simple algebraic summations. That is to say, these others seem to assert that a poor demand would be compensated for by a good hypothesis, a poor skill by a strong differentiation, a poor differentiation by a strong appetite, and the like. Indeed it seems to me that *all* the associationistic psychologies, whether they be of the trial-and-error variety or of the conditioned reflex variety really imply just such simple algebraic summations. What I have distinguished as 'demands,' 'appetites,' 'differentiations,' 'skills,' 'hypotheses,' and 'biases' the associationistic psychologies

¹⁰ See also the problems concerning this f_s function between $\Sigma(OBO)$ and hypotheses already discussed above.

have lumped together, one and all, as mere *S-R*'s. If the rat be very hungry (have a strong demand) this, for them, is but an enhancement of some *S-R* connection; if he have a strong appetite as a result of the type of goal presented, this also is but some *S-R*, stronger than it otherwise would have been; if the given maze-bifurcation present lots of stimuli (leads to clear differentiations) again, merely some *S-R*'s are stronger; if the maze be constructed to require unusual motor skill from the animal, this again means merely a strengthening (or in this case probably a weakening) of some bond or other; if $\Sigma(OBO)$ has become large—if, that is to say, the hypotheses have become 'developed and sure' this also means but better *S-R* connections; and finally, if the maze be shaped to induce, say, a strong centrifugal swing to the right or a strong forward-going tendency to the left, this, also is for them, but a matter of the strengthening of one or another *S-R* bond. And the final value of the resultant behavior-ratio is then obtained by all such psychologies by a simple toting up of these plus and minus, strong and weak, *S-R* bonds. But I am very doubtful of the adequacy of any such simple type of additions.

Let me recall again the facts of 'latent learning.' During latent learning the rat is building up a 'condition' in himself, which I have designated as a set of 'hypotheses,' and this condition—these hypotheses—do not then and there show in his behavior. *S*'s are presented, but the corresponding *R*'s do not function. It is only later, after a goal has been introduced which results in a strong appetite, that the *R*'s, or as I would prefer to say, the *B*'s, appropriate to these built-up hypotheses appear. So long as there is no appetite for what is found at the end of the maze, strong demands, plus strong hypotheses do not add up at all. A strong hypothesis and a strong demand do not compensate for a weak appetite. And a strong demand and a strong appetite cannot in their turn overcome a weak hypothesis. And so on. The ways of combination of the intervening variables do not seem those of simple scala addition.

Or consider, as another example, the addition of two

hypotheses. And suppose that instead of the usual two-way choice-point, we had one such as that shown in Fig. 16. In this set-up after a long series of preliminary training in which only the two side-paths were open, the middle path was also

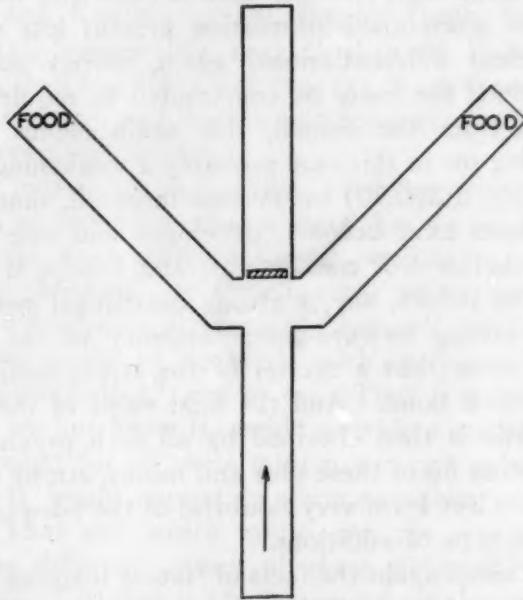


FIG. 16

opened up (I refer here to an actual experiment devised and carried out at California by Mr. R. S. Crutchfield). As a result of the preliminary training the two hypotheses of food to the left and food to the right were built up. It appeared, however, in the test runs, that these then added together in such a way as to make a very strong resultant tendency to go straight ahead when the third central path was opened—in short, a very much stronger tendency to go ahead than was found to have resulted from the two hypotheses which got built up when in another set-up the two side paths were as shown in Fig. 17. The laws of the addition of hypotheses here appeared, in short, not as scalar and algebraic, but as vectorial.

Or, again consider the facts of rat behavior which ordi-

narily go under the names of 'insight' and reasoning, that is to say, such facts as have been gathered by Honzik and myself (128, 47) and by Maier (74, 75, 76, 77).¹¹ These are again, as

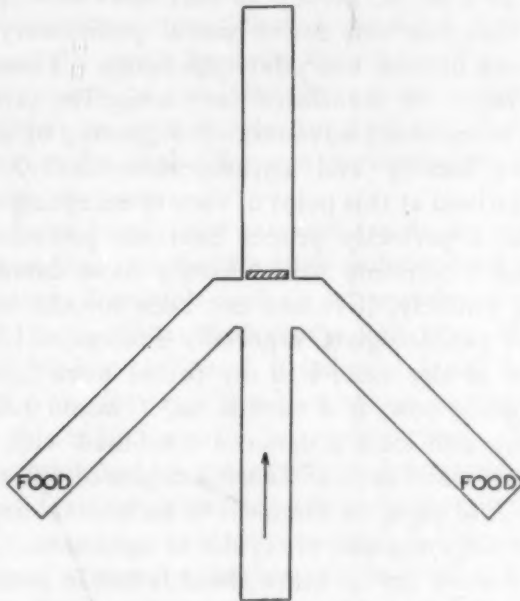


FIG. 17

I see it, also primarily facts concerning the addition of hypotheses. The addition here also is anything but simple and algebraic.

And so I am brought finally to my present confession of faith—namely, that Professor Lewin's topological and dynamic concepts (65, 66) now seem to me the best lead that I have at present for conceiving the nature of this f_3 function. I neither understand nor approve them in their entirety. And, if I were clever enough, I should undoubtedly try in many ways to improve upon them. But nonetheless, even as they are, they seem to me by far the most stimulating and important ideas which have appeared in psychology (that is, in pure psychology, as distinct from physiology or embryology) in the past decade.

¹¹ For a summary of most of these facts see Munn (97, Chap. VII) and Heron (40).

One final point, concerning my thinking about the f_s function. I am at present being openly and consciously just as anthropomorphic about it as I please. For, to be anthropomorphic is, as I see it, merely to cast one's concepts into a mold such that one can derive useful preliminary hunches from one's own human, everyday experience. These hunches may then, later, be translated into objective terms. But there seems to me every advantage in *beginning* by conceiving the situation loosely and anthropomorphically. I might never have arrived at this point of view of accepting anthropomorphism as a perfectly proper heuristic procedure all by myself. And I certainly would hardly have dared advance such a view publicly, if it had not been for the counsels of several other psychologists, especially Professors Liddell and Zener. But, in any case, I in my future work intend to go ahead imagining how, *if I were a rat*, I would behave as a result of such and such a demand combined with such and such an appetite and such and such a degree of differentiation; and so on. And then, on the basis of such imaginings, I shall try to figure out some sort of f_s rules or equations. And then eventually I shall try to state these latter in some kind of objective and respectable sounding terms such as vectors, valences, barriers, and the like (to be borrowed for the most part from Professor Lewin).

Also, of course, I shall try to do experiments similar to those of Lewin and his students in which these intervening variables (as extrapolated from their correlative independent variables) are given such and such supposed values and then the final behavioral outcomes measured.¹²

But many of you must have been asking yourselves all this time: what about the H , A , T , and E variables? In the defining experiments I have suggested so far, which have been concerned primarily with the environmental variables, these 'individual difference variables' are assumed to have been given average standard values. We rat-workers have

¹² As a beginning in this direction we already have some rat experiments by Hall (34, 35, 36) and Hall and Ballachey (37), and a recent set of experiments by Wright (148), but the latter were done unfortunately, from my point of view, not upon rats but upon children. But analogous experiments could, I believe, be done with rats.

always done this, perhaps unconsciously. We have tried to keep heredity normal by using large groups, age normal by using rats between 90 and 120 days old, previous training normal by using fresh rats in each new experiment, and endocrine and nutritional conditions normal by avoiding special dosages and also again by using large groups.

But suppose, now, our interests *be* in individual differences, *per se*. What experiments do we carry out then? It seems to me that individual-difference psychologists here tend to do two sorts of things.

On the one hand, they attempt (as do we environmental psychologists) to manipulate their independent variables for whole groups of animals and to get correlated variations in $B_L/(B_L + B_R)$. Thus they vary heredity, H , as Tryon (129) and Heron (41), and Rundquist (104) have done in controlled ways for large groups and get corresponding variations in this behavior-ratio, for such groups. Or, they vary age, A , as Stone and his students have done (114), also for large groups and again get corresponding variations in the behavior-ratio. Or, they vary previous training, T , that is, they study the effects of transfer—and here we have all taken pot shots—the first important experiment was, perhaps, that of Webb (142) and the last seems to be that of Bunch and Rogers (15)—and again attempt to get corresponding variations in the behavior-ratio. Or, finally, they vary drugs, endocrines and vitamins, E , and get correlated variations in $B_L/(B_L + B_R)$. Here there are too many experiments for me to attempt to list them.¹³

Secondly, however, the individual difference psychologists have also done another *more characteristic* type of experiment. They have accepted from God, and from the accidents of miscegenation and of nursery schools, very large heterogeneous samples of rats and then they have put each such sample

¹³ One of the best known early experiments was that of Anderson and Smith (1) on the effect of insufficient diets. And recent further important experiments on diet are those of Maurer (78, 79, 80), and Maurer and Tasi (82, 83), Bernhardt (7), Muenzinger and the Poes (96, 98, 99). For recent important experiments on drugs, see Miller and Miles (85) and Williams and O'Brien (144). Also for a summary, see Moss's own chapter in 'Comparative Psychology' (86).

through a miscellaneous assortment of experiments (*i.e.*, the different types of mazes that, in American rat-culture, are required of young rats in school, and also the different types of maze, discrimination-box, food, times since eating, and the like, which are required of old rats in polite society); and then they have obtained correlations and worked out factor analyses. And, finally, these individual-difference psychologists have ended up with their notions concerning the number and nature of the fundamental traits or capacities—'The Vectors of Mind' (120). These traits or capacities are, of course, but a new type of intervening variable and it would be nice, for me, if they fitted in neatly with the sort of intervening variables already suggested. They could then be put into my diagram as shown in Fig. 18. But, alas, at present

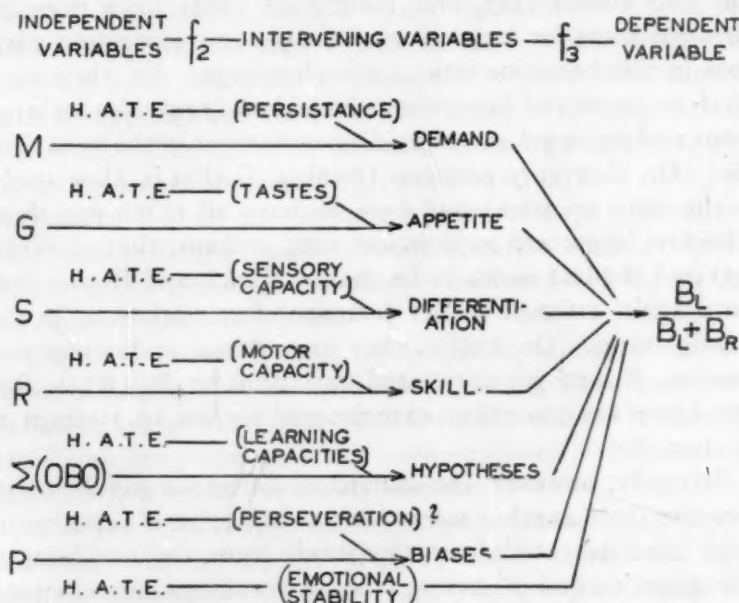


FIG. 18

the results of factor analysis do not seem to suggest any such simple or agreed-upon results. You all know how the controversy rages from Spearman's one or two factors (109) through Kelley's (52) and Thurstone's (120) three to nine

factors, differing somewhat in each set-up¹⁴ to Thorndike's (116, 117) and Tryon's (131)—God only knows how many.¹⁵

By way of conclusion, I want now, however, to turn to one wholly new point. I want to suggest that there also appear in maze behavior types of activity other than the simple B_L 's and B_R 's which we have thus far talked about. If these latter be called 'achievement behaviors,' then these new types of activity which I now have in mind, may be called 'catalyzing behaviors.' And it seems that we rat psychologists have to date rather pigheadedly (*i.e.*, like Professor Liddell's pigs) ignored such catalyzing behaviors.

I have two instances which I would here like to call to your attention, although I believe that in the future technological advances in recording will bring to the fore many others for study. The first of these two examples consists of those 'lookings or runnings back and forth' which often appear at the choice-point and which all rat-runners have noted, but few have paid further attention to. And the second type is that disrupted sort of activity which appears when a previously obtained goal object is removed or blocked. Let me begin with the former.

A few years ago (121; 122, Chap. XIII) I had the temerity to suggest that such 'lookings back and forth' might be taken as a behavioristic definition of *conscious awareness*. This was, no doubt, a silly idea. I would hardly dare propose it now. But, at any rate, such behavior is interesting and deserving of further study. Anthropomorphically speaking, it appears to be a 'looking before you leap' sort of affair. Klüver (53) and Gellerman (29) have recorded it in connection with the behavior of monkeys, chimpanzees and children. And, further, I have recently learned that Professor Muenzinger and his students have also been keeping records of it in rats and that they have called it 'vicarious trial and error'—or, more briefly, *VTE*. I shall, therefore, designate such behavior as *VTE* or B_{VTE} from here on.

¹⁴ I think here of Vaughn's recent important monograph (120) in which he finds eight factors governing maze behavior.

¹⁵ For a general discussion of the problem of individual differences in animals see, also, Tryon (130).

First, let me show you some individual rat curves obtained by Dr. Evelyn Gentry (94) in Muenzinger's laboratory. The one rat had a difficult discrimination—namely, to go left when a tone is sounded; the other had an easy discrimination—to go towards the white in a white-black discrimination box (Fig. 19).

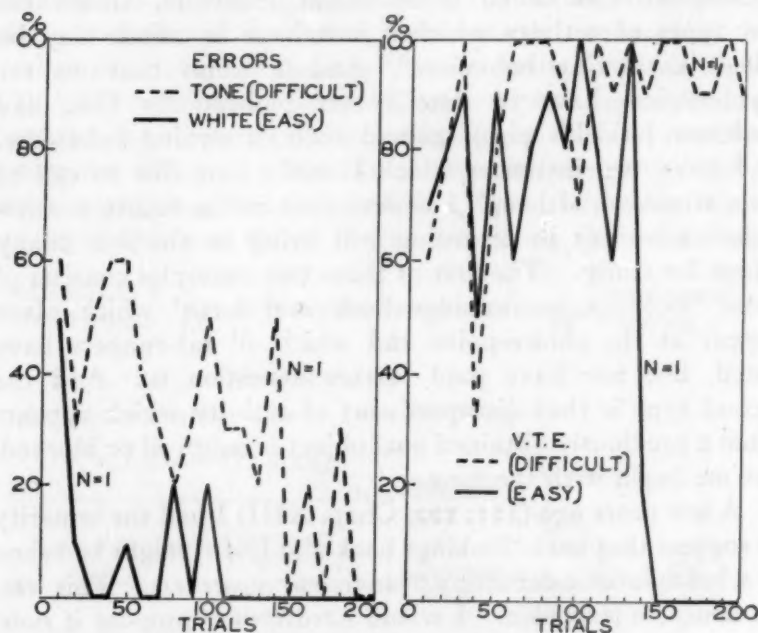


FIG. 19

At the left are the error curves and at the right the *VTE* curves. Whenever the rat looked one or more times before making his overt choice in a given trial that trial was recorded as having involved a *VTE*. The points on the curves are averages for ten trials. The solid curves are for the easy discrimination and the dash curves are for the difficult discrimination. As you see, there tended to be more *VTE* and the latter persisted longer for the difficult discrimination than for the easy one.

Next, let me present some recent data on *VTE* obtained by Mr. M. F. Friedman at California on the effect of moderate

amounts of cortical lesion ¹⁶ (see Fig. 20). The problem was learning to turn left on a simple elevated *T* where one arm led to food and the other did not. The dash curves are for the brain lesion group and the solid curves are for the control

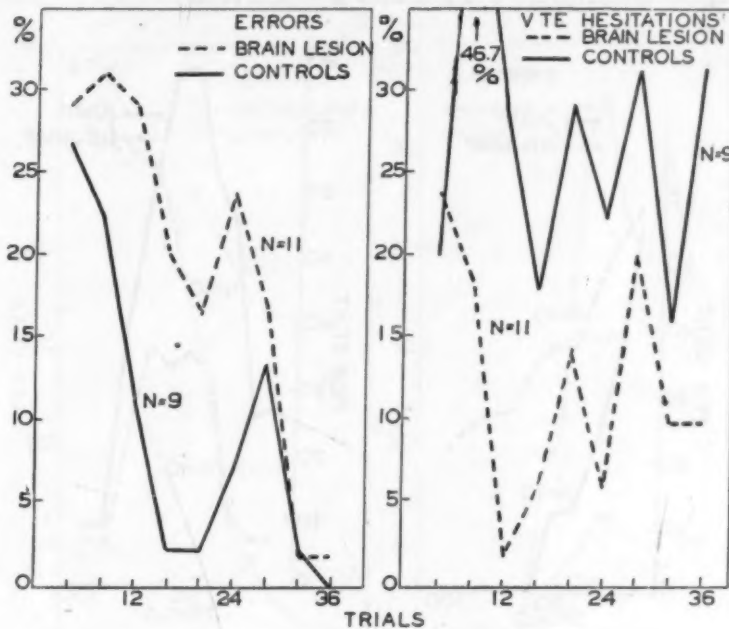


FIG. 20

group. Each point is an average of 4 trials. The normal animals exhibited more *VTE* and learned faster than did those with cerebral insults.

Next, I present some curves obtained by Honzik with an elevated discrimination set-up. The animal had to discriminate between a black and a white face-on door. There was a partition projecting out between the doors. White was the positive stimulus. One group ran over a continuous platform and could run back around the projecting partition if they chose the wrong door first. The other group had to jump a gap of $8\frac{1}{2}$ inches to a 4-inch ledge just in front of the doors. If this jump group chose incorrectly, they had to

¹⁶ The histology necessary for determining the actual amounts of these lesions has not yet been done.

jump back again to the starting platform and then make a second jump to the correct door (Fig. 21). The solid curves are for the jump group and the dash curves for the non-jump group. Each point represents an average of 10 trials. The jumpers made more *VTE*'s and learned faster.

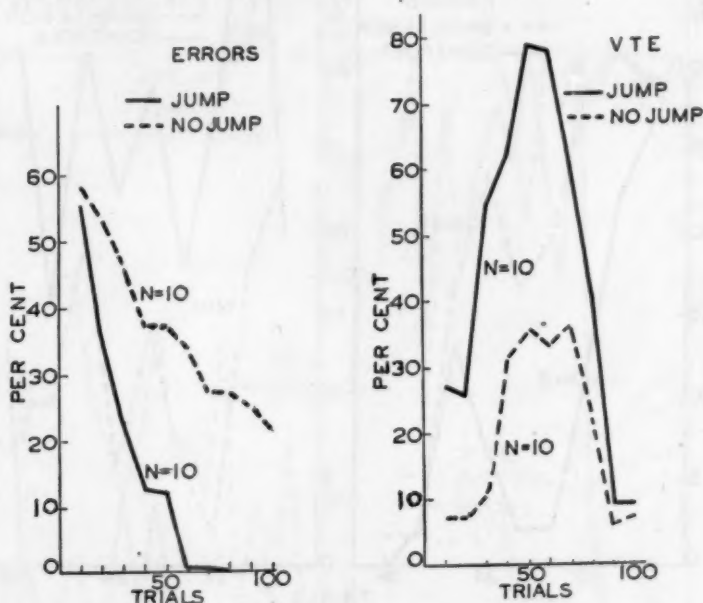


FIG. 21

Finally, let me present a set of curves also obtained by Honzik, in a similar set-up, but for two different jump-groups (Fig. 22). The conditions for the one-jump group were those just described. We may call them here the near-jump group. For the others, which we may call the far-jump group, the farther side of the gap was fifteen inches from the to-be-discriminated doors and the taking off platform $23\frac{1}{2}$ inches from these doors. Solid curves are for the near jumpers, dash curves for the far-jumpers. Each point represents an average of 10 trials.

The near-jump group learned faster and exhibited more *VTE* than did the far-jump group. It is to be noted that the far-jump group probably could not see the differences between

the two doors at the place of "taking off" very well. Hence their poor error score. Further, because they could not see very well, it did them little good to 'go in for' 'looking before they leapt.' And, in fact, the *VTE*'s for this far-jump group were decidedly less than for the near-jump group.

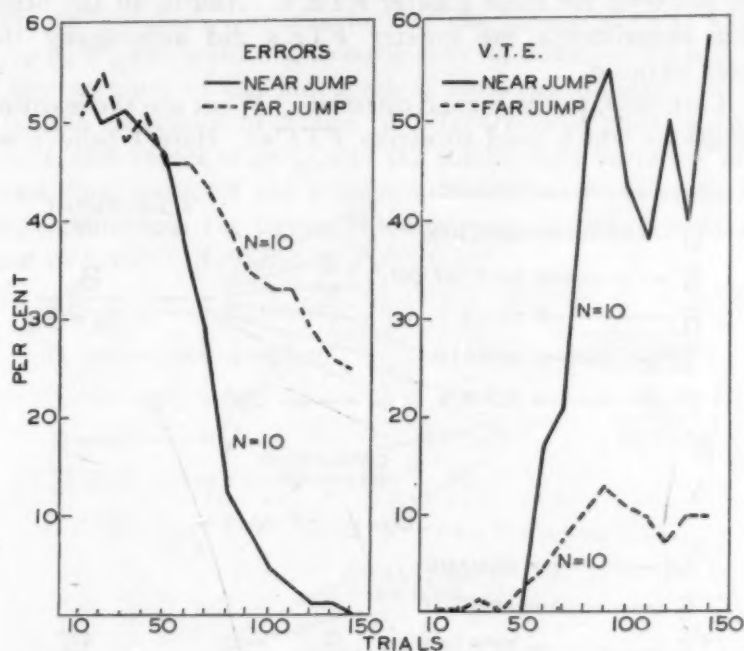


FIG. 22

Let me briefly summarize: (1) For a difficult discrimination such as learning to turn left when a tone is sounded there was slower learning but more *VTE* than for an easy, white-black discrimination; (2) On a simple *T*, normal rats showed faster learning and exhibited more *VTE* than did brain lesion rats; (3) With a near-jump, jump rats learned faster and showed more *VTE* than did non-jump rats; (4) Near-jump rats learned faster and exhibited more *VTE* than did far-jump rats.

What, now, is to be our theoretical envisagement? Obviously, the question divides into two: (1) what effect do *VTE*'s, when evoked, have upon learning; (2) what are the conditions of learning which favor the evoking of such *VTE*'s?

In answer to the first question I shall postulate that *VTE*'s always aid the learning which they accompany. In the sole case, that of the difficult discrimination, where the poorer learning was accompanied by more *VTE*'s I believe that this learning was nonetheless faster than it would have been if it had not been for these greater *VTE*'s. And in all the other three experiments the greater *VTE*'s did accompany the faster learning.

Turn now, to the second question. What are the learning conditions which tend to evoke *VTE*'s? Here I believe we

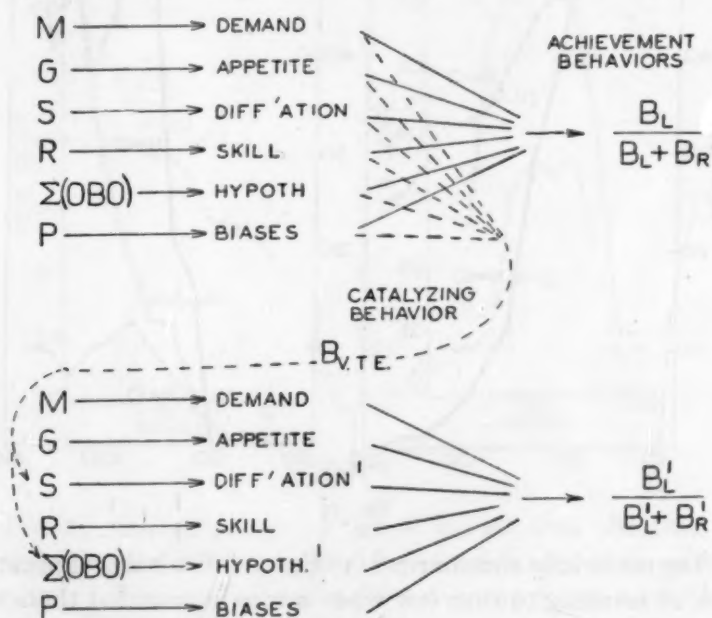


FIG. 23

are not yet ready for any general answer. I shall therefore merely re-enumerate for your benefit the conditions of the four experiments. The conditions favorable to *VTE*'s in these experiments were: (1) a difficult discrimination; (2) a normal brain; (3) a gap to be jumped which induced caution, and (4) a nearness of the jumping platform such that the extra caution exposed the animal longer to the critical stimuli.

Finally, let me by another figure suggest how I would

propose to fit this catalyzing *VTE* behavior into my general causal diagram (Fig. 23). You will note that I have shown the *VTE* behavior—symbolized as B_{VTE} —as an auxiliary result of the ‘intervening variables.’ These latter are to be conceived as tending to produce their usual ‘achievement behavior’ $B_L/(B_L + B_R)$. But, in addition, they produce more or less B_{VTE} , and the further catalyzing effect of such B_{VTE} is, I have assumed, in some way to enhance the values of one or more of the independent variables themselves—in this case especially of S and of $\Sigma(OBO)$ —and thus to help induce new values of certain of the intervening variables and a new final value of the achievement behavior. That is to say, as shown in the figure, the achievement behavior takes some new value $B'_L/(B'_L + B'_R)$.

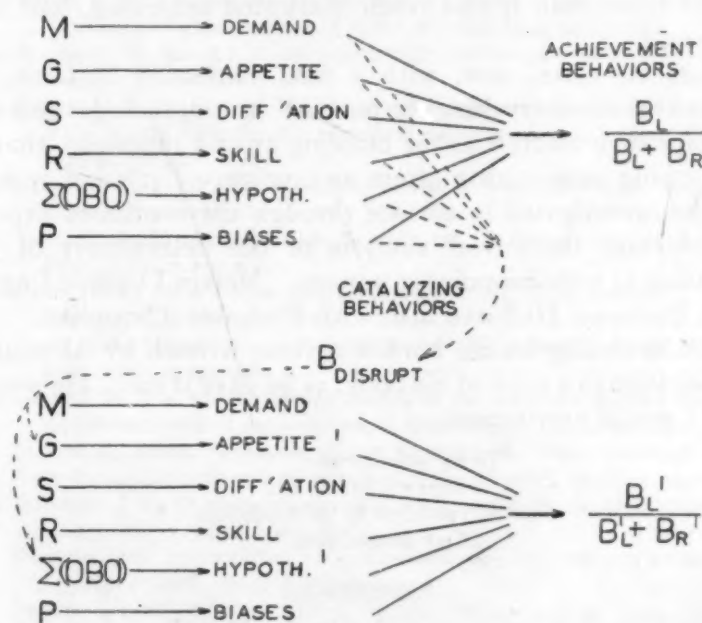


FIG. 24

Turn now briefly to the case of the disruption behavior which occurs when an expected goal is not obtained. I have as yet no curves or detailed data concerning either the causes or the results of such disruption behaviors. I believe, how-

ever, that they also are to be conceived as auxiliary, catalyzing sorts of affair which react back upon the independent variables and make the final values of the resultant behavior-ratios different from what the latter originally would have been.

The rat's disrupted behavior is a surprised sort of hunting about and exploring. And it is my contention as shown in Fig. 24 that this surprised hunting and exploring brings about new values of the independent variables—especially of G and $\Sigma(OBO)$,—and thus causes a different outcome in the final behavior ratio. The disrupted behavior enhances a new negative aspect in what was originally a positive goal. In short, I am assuming that because of this disrupted searching the rats are better in the next trials about not continuing to go to that side where the goal has been blocked than they would have been if this, their disrupted searching, had not appeared.

Let me close, now, with a final confession of faith. I believe that everything important in psychology (except perhaps such matters as the building up of a super-ego, that is everything save such matters as involve society and words) can be investigated in essence through the continued experimental and theoretical analysis of the determiners of rat behavior at a choice-point in a maze. Herein I believe I agree with Professor Hull and also with Professor Thorndike.

So in closing let me borrow a verse written by Alexander Meiklejohn in a copy of his book, as he gave it me. He wrote, and I would now repeat:

"To my ratiocinations
I hope you will be kind
As you follow up the wanderings
Of my amazed mind."

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THE THALAMUS AND EMOTION

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For many years some relation between the thalamus¹ and emotions has been suspected. As early as 1822 Fodéra reported that stimulation of a region between the level of the *chiasma* and the *corpora quadrigemina* induced cries and other emotional expressions which could not be elicited by stimulation of the cerebral hemispheres. Bechterew (3, 4, 6) was the first to collect evidence systematically to show that the thalamus is concerned in emotional expression. He described the production of a variety of expressive and visceral responses by stimulation of the thalamus, the expression of major emotions in the decorticate animal, and the appearance of spasmodic laughter and weeping after lesions involving the internal capsule in man (3). His objective system of psychology had no place for a separate category of emotions. He developed the Darwinian concept of the origin and adaptive character of expressive and visceral reactions, but made no effort to relate them to emotional experience (5).

From his work on the sensory pathways, Head (18) was led to postulate a thalamic center concerned with the affective character of somatic sensation. He assumed that damage to the center or to its corticopetal connections produced hypaesthesia and that release of the center from cortical control resulted in intensification of the affective character of the sensations. He was not explicit concerning the mode of action of the thalamic center. He seems to have accepted the James-Lange theory for the emotions, yet to ascribe a direct action on the affective value of sensations of pain and tickle to a thalamic center.

¹ In the following discussion I shall use the term thalamus to identify the general region of the diencephalon. This is in accord with frequent usage, and more accurate designation of the various regions of the diencephalon is immaterial to the argument.

Increasing doubt of the James-Lange theory of emotions has led more recently to a search for an alternative physiological hypothesis. Of the various ones which have been proposed, level of central tonus, conflict of reaction tendencies, and the like, an elaboration of Head's theory of thalamic function has excited the widest interest and following. It has been expressed with some variations by Dana (12), Cannon (10), Bard (1, 2), and Harlow and Stagner (17).

The theory of emotion as a function of thalamic activity has been most precisely stated by Cannon (10) in essentially the following form. Afferent impulses initiated by the stimuli capable of arousing emotion are transmitted to the cortex. Within the cortex they are integrated to arouse the appropriate overt behavior. A center in the thalamus is also excited, either directly as the sensory impulses are relayed there, or secondarily by impulses from the cortex. The thalamic center (center for emotion) discharges somewhat explosively, exciting the effectors in patterns which constitute the 'expression' of the emotion and also discharging to the cortex, where the impulses from the thalamic center add the 'peculiar quality of emotion' to the simple sensation aroused by the direct effects of the exciting stimulus. In addition it is suggested that the flood of impulses from the thalamus constitutes the emotional tension and accounts for the dynamic or motivational character of the emotion.

Harlow and Stagner (17) have assumed that only one type of facilitation, corresponding to a general excitement, is contributed by the thalamus and that differentiation of diverse emotions is the result of cortical discriminative processes depending upon the stimulating situation. Bard (1, 323-324) points out, in criticism of these writers, that the motor patterns elicited from the thalamus are specific for different emotions and consequently imply a difference also in the pattern of corticopetal impulses from the thalamic center. He thus seems to hold that the thalamic center determines the qualitative differences between emotions.

To account for the phenomena in certain pathological cases the theory further assumes that the cortex has both excitatory

and inhibitory influences upon the thalamus and that the inhibition is effective both upon the motor and the corticopetal excitations arising in the thalamic center.

The theory has the virtues of clearness and simplicity and of apparent support by a large body of experimental evidence. In these respects it has an advantage over vague theories of nervous tension and over more complicated formulations which seek to deal with the intellectual and motivational aspects of the problem. From the psychological standpoint, however, it is inadequate in several respects. Like the James-Lange theory, which it seeks to displace, the thalamic theory is concerned primarily with the problem of experience. It minimizes, if it does not disregard entirely, the problem of motivation which looms so large with the development of dynamic psychology and psychopathology. Further, a unity and constancy of emotional experience is assumed, which is scarcely justified by introspective findings. It is by no means established that there are any identifiable emotions which have a constant qualitative character, or that the 'peculiar quality of emotion' is a genuine phenomenon. The evidence which has been cited for the specificity of different emotions is the constancy of the patterns of bodily reaction (2), yet the proponents of the thalamic theory have denied subjective significance to these very reactions in their attack upon the James-Lange theory.

These criticisms, however, are not serious obstacles to the thalamic theory of the emotions. It could doubtless be elaborated to provide a basis for motivation and it has been modified by Harlow and Stagner to conform to psychological data concerning the importance of intellectual factors in the subject's identification of his emotions.

A far more fundamental issue for the theory is that of the validity of the evidence upon which it is based. The theory ascribes three functions specifically to the thalamic region: action as a higher motor center in the integration of patterns of expressive reaction, the initiation or facilitation of nervous impulses which modify cortical processes to give them an emotional character, and the reinforcement of behavior in the

sense of the addition of an emotional drive. In this paper I propose to examine the evidence that the thalamus contains a specific center or centers for these functions.

THE INTEGRATION OF MOTOR PATTERNS IN THE THALAMUS

Bechterew (3, 5) compiled evidence of the functions of the thalamus in the elaboration of expressive movement. On electrical stimulation of the thalamus he was able to elicit vocalization, respiratory and circulatory changes, erection of hair and other expressive movements from a variety of animals. T. G. Brown (8) described respiratory movements resembling laughter on stimulation of a region medial to the red nucleus and sighing on stimulation of the caudal portion of the thalamus of the chimpanzee. Other investigators have added to the number of visceral activities found to be elicitable from this region.

Bechterew (3) also reported that decorticate animals were readily induced to display patterns of expressive movement but that after section behind the thalamus only partial patterns, cries and the like, could be elicited, and these only by strong stimulation. The careful systematic studies of Bard (1, 2) have confirmed these observations on thalamic preparations and have localized the motor center for some of the expressive movements more closely in the caudal part of the hypothalamus.

These studies leave no doubt that there are centers within the thalamus whose excitation elicits organized patterns of emotional expression. Parts of the patterns are integrated at lower levels. Elements of the fear and rage patterns may be elicited from midbrain preparations (Bechterew, 3; Woodworth and Sherrington, 32), and stretching, which might be interpreted as a sign of contentment, may occasionally be elicited even from the spinal animal, but for complete integration the caudal portion of the thalamus is essential. The occurrence of spasmodic laughter and weeping in cases of diplegia, together with Graham Brown's observation on the chimpanzee, tend to localize the motor centers for these expressive patterns also in the thalamic region.

In the hierarchy of motor centers we may then recognize the thalamic region, especially the hypothalamus, as the region within which the complex patterns of expressive movement are elaborated. It does not follow from this, however, that the pathological phenomena of hyperexcitability of emotional reactions are due solely to release from cortical inhibition or that the thalamic motor center for expressive movement contributes to the emotional experience.

Excitability of the motor centers.—All investigators have reported a more ready elicitation of some emotional expressive movements from the decorticate than from the normal animal. In carnivora these have been described as rage and fear. In the anencephalic infant startle, expression of pain, and crying are readily obtained. Comparable data are not available for the human adult, but the spasmodic laughter and weeping of pseudobulbar paralysis and the excitement and euphoria of frontal lobe cases have been interpreted as representing a similar condition. On the basis of Hughlings Jackson's principle that lesions cannot produce a gain in function, the increased reactivity must be ascribed to a decrease in inhibition. Head (18) developed this conception to explain the hyperalgesias resulting from thalamic lesions, assuming that the release of a thalamic affective center from cortical inhibition leads to an increase in affective discharge to the cortex. The theory of thalamic function in emotion has made use of the same concept to account for the increased emotional excitability in organic nervous disorders.

Although we may assume that the increased excitability of the motor centers is a result of withdrawal of inhibition, a survey of the evidence leaves some doubt as to the source of this inhibition in the normal animal. It is by no means certainly established that the disturbances of emotional expression in pseudobulbar palsy are actually due to the interruption of cortico-thalamic paths. Wilson (31) reported spasmodic laughter and weeping in progressive lenticular degeneration, which involves the striatum, but perhaps not important cortico-thalamic tracts. Tilney and Morrison (30) have reviewed the cases of pseudobulbar palsy for which there

were anatomical controls. Of these, half which exhibited spasmodic laughter and weeping had no lesions in the striatum or thalamus, and half of the cases with lesions in these structures did not show disturbances of expressive movement. The ascription of the loss of control of emotional expression in such cases to interruption of cortico-thalamic inhibitory fibers is an inference from the thalamic theory and is not based upon conclusive anatomic evidence.

Facile laughing and weeping, especially the latter, appear in a number of other conditions, such as extreme fatigue, debility after infectious disease, depressions of pregnancy and menopause, hysteria, and under the effects of various drugs. Since these conditions seem to form a continuous series with the extreme condition in pseudobulbar palsy, and since to assume that all of these conditions are forms of partial decortication is to beg the question, we have little reason to believe that convulsive laughter and weeping are due primarily to the interruption of cortico-thalamic inhibitory tracts.

There is no other convincing clinical evidence of the release of emotional expression by any form of cortical lesion. The frontal lobe symptoms of excitement or euphoria (14) may be so interpreted, but they may also be explained as positive reactions or excitation, due to a lower level of comprehension of social situations. In any event there is no evidence that release of the thalamus rather than of other cortical regions from frontal inhibition is responsible for them. The phenomena are simply too complex to serve as evidence for any theory.

Thus we see that, although normal inhibition of the thalamic centers for expressive movement is indicated, there is some uncertainty as to the source of the inhibition.

The rage response is more readily elicited and is more violent in the decorticate than in the normal animal and this justifies the postulation of a normal inhibition of the rage reaction by the cortex. But it is quite possible that variations in the excitability of other emotional reactions may be conditioned in entirely different ways and it is still unsafe to generalize from the condition of the decerebrate animal to the various clinical pictures of emotional hyperexcitability.

The motor centers and emotional experience.—The only direct evidence concerning the discharge to the cortex from the thalamic motor centers for emotional expression is that derived from cases of spasmodic laughter and weeping. Dana (12), Wilson (31) and others have pointed out that many patients with these symptoms disclaim emotions appropriate to the expression,² and Dana, Wilson, Cannon (10) and Bard (2) have cited such dissociation of emotional experience from expression as evidence against the James-Lange theory. It is to the same extent, however, evidence that discharge to the cortex from the thalamic motor centers for emotional expression is not the basis of the subjective quality of emotion. For the motor discharge is specific in different emotional expressions, as Bard (2) has pointed out, and we have no basis for assuming that the center can discharge one emotional pattern to the effectors and another to the cortex. We must therefore recognize on the basis of these clinical cases that the thalamic motor centers, which are concerned with the elaboration of emotional expression, are not the source of corticopetal impulses which determine the subjective character of emotion.

To preserve the thalamic theory this forces the postulation of another thalamic nucleus which contributes the emotional quality and may be dissociated from the motor nuclei. But this assumption immediately involves further difficulties. To contribute emotional excitation to the cortex, the nucleus must be excited, either by sensory impulses relayed in the thalamus or by descending impulses from the cortex. It must be dissociated from the thalamic motor nuclei by the same lesions which free the latter from cortical inhibition, since it still contributes appropriate emotions when the action of the motor nuclei is inappropriate. It must be excited by other sensory impulses or cortical processes than those which activate the motor nuclei, since the 'emotional' and motor nuclei can be aroused to opposite activities by the same

² In one patient of this type, whom I have studied, spasmodic laughter could be induced by reference to his very distressing home situation. He not only denied amusement but claimed that he felt very sad and depressed during the spasm of laughter. This is evidence that there may be an intense emotional experience which is the opposite of that represented by the thalamic discharge.

stimulus. We seem to be approaching here the extravagances of the diagrammatic theories of aphasia, and I shall not pursue the matter further.

Before such speculations are justified, it must be demonstrated that the thalamus does contribute something to emotional experience. The only positive evidence which has been adduced in support of the origin of centripetal affective or emotional impulses within the thalamus is that presented by Head in his studies of pathological changes in somesthetic sensation. He reported changes in the character of somesthetic sensations following thalamic lesions, which he interpreted as affective disturbances due to interference with the functions of a specific thalamic center. In the following discussion I shall attempt to show that the symptoms reported by him are not due to disturbance of affect and that they are not confined to lesions involving the thalamus and its cortical connections.

AFFECTIVE REINFORCEMENT OF SENSORY IMPULSES BY THE THALAMUS

The facts from which Head deduced his theory of thalamic function were the following symptoms in cases of thalamic lesion:

1. Pricking, scratching, heat, pressure, or continued stroking may be felt by the patient as intolerably disagreeable or painful upon the affected side when like stimuli on the normal side have no such disagreeable character.
2. In other patients stimuli which are felt as painful on the normal side are not felt as painful on the affected side, although their pricking, scratching, or other qualitative character is recognized.
3. In hyperalgesia the absolute threshold is not lowered, but the emotional effects of the adequate stimulus are heightened.
4. Warmth and, rarely, tickling may be felt as abnormally pleasant on the affected side.
5. In a few patients showing increased affectivity of pain, auditory stimuli, especially those of an emotional character, produce tingling and other unpleasant sensations in the affected parts of the body.

To account for these facts Head assumed that there is a center in the thalamus which adds the affective character to the somesthetic sensations. Hyperalgesia and increased pleasantness of warmth were interpreted as due to release of this center from cortical inhibition, hypoalgesia to interference with the activities of the center, or with its afferent path to the cortex.

We must raise two questions concerning this interpretation: Are the 'affective' changes specifically dependent on the thalamus? Are the facts relevant to the problem of emotion?

Pathological 'affective' changes restricted to somesthetic sensations.—We may first dismiss briefly the claim that the thalamic lesions involve a general change in affective experience. The evidence advanced for this was the report of emotional disturbance by music, of which Head records two cases. These cases have been cited by Bard (2) as evidence that "the feeling tone of any sensation is a product of thalamic activity." But what Head actually reported was that the music caused unpleasant somesthetic sensations. "One of our patients was unable to go to his place of worship, because he 'could not stand the hymns on his affected side' and his son noticed that during the singing his father constantly rubbed the affected hand" (18, p. 560). Again, "As soon as the choir began to sing, a 'horrid feeling came on the affected side, and the leg was screwed up and began to shake.'"³ In no case was the affect referred to the source of emotional stimulation, to the music, but always to sensations of somatic reaction to the stimulus. Only one interpretation of the reports is possible; that the emotional stimuli gave rise to expressive reactions which, owing to the hyperaesthesia on the affected side, were felt there more acutely. Nothing more than an abnormality of somesthetic sensation is indicated by the observations; certainly not a general increase in affectivity.

There is, I believe, no case on record of affective disorders

³ The origin of the motor activity exhibited in this case remains entirely obscure. It might be interpreted as an excessive discharge of 'emotionally expressive' movement to the leg, implying release of an emotional center from inhibition, but might also be a secondary reaction due to the greater intensity of somesthetic impulses from the affected side.

involving other sense modalities in a manner similar to that described for somesthesia. Photophobia occurs in certain retinal conditions and in migraine but not after central nervous lesions, and it involves pain on photic stimulation, not an affective reaction to light. Disagreeable olfactory hallucinations are frequent and may have a sensory basis, but they result from lesions within the olfactory system and I have not seen an account of any change in the affective character of olfactory sensations from lesions in the thalamus or internal capsule. Certainly no such affective changes were reported for Head's cases. It is noteworthy also that sensations of posture showed no alteration in affective character in his cases.

The release of an affective or emotional center in the thalamus from cortical or other inhibition should result in a change in the affective value of all stimuli capable of arousing emotion, if the region is to be regarded as a general center of affect or of emotion. Such a general change in the level of affect does not occur after thalamic lesions. The affective changes produced are restricted to the narrow group of somesthetic sensations, pain, heat, tickle, and warmth. The clinical evidence cannot be cited legitimately as bearing upon the general problem of affectivity or emotion.

The phenomena are peculiar to a single limited group of sense modalities. Since these are not the only sensory impulses relayed in the thalamus and since the sense modalities included in the group are peculiarly related in several ways, we must inquire whether the affective changes are a property of the thalamus or of some characteristic of these special modalities.

The locus of somesthetic 'affect.'—Hyperalgesia may result from lesions anywhere along the conduction path from the end organ to the thalamus.⁴ Rivers and Head (27) reported an excessive painfulness of pain stimulation during regeneration of cutaneous nerve and this fact has been confirmed by more recent investigators. Hyperalgesia occurs in neuritis

⁴A number of pertinent cases have been reported by Davison and Schick (Proc. Asso. Res. Nerv. Ment. Dis., 1935, 15, 457-496).

and other diseases of peripheral nerve. It has been ascribed to irritative lesions but persists in cases of long standing where active irritative processes are improbable.

It is also of common occurrence in lesions or diseases of the cord. In syringomyelia it has been ascribed to irritation, but it is noteworthy that in such cases there is no evidence of motor irritation. After traumatic destruction of parts of the cord hyperaesthesia and hyperalgesia may be pronounced. In Brown-Séquard paralysis, after hemisection of the cord, it is a characteristic feature (7). Kocher (22) lists ipsilateral hyperaesthesia for touch, pain, and sometimes heat and cold as characteristic symptoms of hemisection of the human cord. Hyperalgesia has been described in numerous experiments with animals. Most of these experiments were acute, but some of Martinotti's animals (24) were apparently kept beyond the irritative stage. Head (18) recognized the existence of hyperalgesia in spinal lesions, although he did not report it in the cases which he studied, and minimized its importance for his theory.

Thus hyperalgesia is not a result only of lesions within the thalamus but may arise from damage anywhere along the afferent path. From the reports one can discover no difference in the character of the 'affective' disturbance corresponding to the site of lesion. The same description of diffuse, burning, intolerable pain is given by all types of patients showing hyperalgesia, whether due to peripheral, spinal, or thalamic lesion. The ascription of a specific 'affective' change to cases of thalamic lesion, different from the condition in spinal and peripheral lesions, is not justified by the published statements of the patients.

Various theories have been advanced to explain the origin of the hyperaesthesias. An early attempt to account for the condition in Brown-Séquard paralysis was that of Kocher (22). He pointed out that the hyperalgesia is more frequent and more severe in cases of complete than of partial transection of the cord. He made the assumptions that the painfulness of pain is determined by summation and irradiation in transmission through the central gray; that in partial hemisection

there is some ipsilateral as well as contralateral conduction of pain; and that in complete hemisection all the pain impulses are relayed to the opposite side of the cord and so caused to irradiate more widely. The theory involves a somewhat teleological conception, that pain impulses blocked from one path must follow another, which is contrary to what we know of the mechanism of conduction.⁵ Nevertheless the emphasis which the theory places upon the factors of irradiation and summation may furnish the clue to the nature of hyperfunction, as we shall see when we examine the characteristics of the sensory impulses.

To account for hyperalgesia after nerve section Head (18) proposed a theory of reduced inhibition. In support of the theory he reported experiments on the glans penis. Stimulation of this region by water of 40 degrees induced severe pain. When the temperature was raised to 45 degrees the sensation changed to one of heat. When the corona as well as the glans was stimulated at this temperature, the sensation again changed to one of pleasurable warmth. From these facts Head deduced that fibers conducting other sense modalities are capable of inhibiting pain impulses. He did not speculate concerning the locus of these inhibitory processes but his theory seems to necessitate the view that the inhibition takes place in the thalamus, since increased affective value of pain or of pleasurable sensations is involved.

In this form the theory encounters several difficulties. If the various types of sensory impulses remained isolated until they reached the thalamus and there discharged into a common pool, there would be reason to ascribe the inhibition to the thalamus. But the same types of sensory dissociation are produced by spinal as by thalamic lesion, though not always in the same combinations. There is therefore no reason to ascribe the mutual influence of sense modalities to the thalamus rather than to other points along the sensory paths, or to assume that inhibitory processes take place there rather than in the spinal gray.

⁵ The Porter phenomenon in the conduction of respiratory impulses does seem to involve just such an all-or-none change in path as was postulated by Kocher (Rosenbleuth and Ortiz, 28).

Lewandowsky (23) states that hyperalgesia from spinal lesions may be present with or without associated defects of other sense modalities. Even admitting, as Head claims, that the measurement of sensitivity by earlier investigators leaves much to be desired, this statement indicates that the degree of hyperalgesia is independent of the severity of other sensory defects. The theory of inhibition would seem, however, to imply that the degree of sensitiveness to pain should be proportional to the amount of other sensory loss. Further, the theory fails to account for hyperaesthesia of spinal origin to tactile stimuli, as it appears in the exaggerated unpleasantness or even the painful character of tickle.

Finally, recent evidence upon the relation of cold, heat, and pain to vasoconstriction and dilatation (Nafe, 26) suggests an entirely different explanation for the results of Head's experiments on the glans and for hyperalgesia after nerve section. If, as Nafe suggests, cold, heat, and pain may be mediated by the same receptors and depend upon the degree of vasomotor tension, the intensity of pain stimulation by heat may be dependent upon the local vasomotor reactions, and not upon any higher central nervous process.

Thus the data presented by Head do not justify the assumption that the 'affective' aspects of somatic sensation are controlled exclusively by the thalamus. Lesions anywhere along the sensory pathway may produce the same 'affective' symptoms and there is no decisive evidence for localizing the phenomena at any specific point along the afferent path.

Common characteristics of somatic sensations which show 'affective' disturbances.—Among the somesthetic sensations those which are concerned with spacial localization do not show any pathological increase in intensity or affectivity. Tactile discrimination and localization of posture may be defective but never become painful or abnormally pleasurable. Only unlocalized touch, pain, pressure, temperature, and tickle become pathologically unpleasant, and warmth and tickle may show the character of increased pleasantness.

All of these have in common a tendency to summation and to collateral irradiation. They are not accurately

localized and when they are of pathological intensity their diffuseness is greatly increased. The normal irradiation of pain is evident at spinal levels in the spread of reflex avoiding reactions and in referred pains. Light tactile stimuli likewise summate at spinal levels, as in the adequate stimulus to the scratch reflex.

Temperature and pressure are closely related to pain and there are recent indications that pain may depend upon summational effects as well as upon specific fibers. Heinbecker, Bishop, and O'Leary (19) have presented evidence that the painful character of pricking stimuli depends upon summation of a number of impulses. Nafe (26) has summarized the evidence that sensations of heat, cold, and pain may originate in different degrees of vasomotor constriction. Gasser (16) suggests that the pressure impulses conducted by large afferent fibers may under some conditions arouse pain.

The above evidence indicates that the painful character of these sensations may be a function of special conditions of summation, rather than of the arousal of special pain fibers or the addition of a specific affective quality, and that what Head interpreted as a specific emotive function of the thalamus is really a phenomenon of summation and irradiation arising from the unique characteristics of conduction of this limited group of sensory paths.

We know little about the conditions underlying warmth, tickle, and the sexual sensations, the so-called pleasurable sensations. They have in common the character of diffuse, poorly localized tingling, and are slow in development and subsidence. In summation and irradiation they resemble pain and differ from other sense modalities. That they are closely interrelated is indicated by studies of the erogenous zones, studies which also suggest a vasomotor element. They cannot be elicited by single stimuli, but must be built up by slow summation. Sexual sensations are sometimes abolished by low unilateral spinal lesions which do not destroy tactile sensitivity of the genitalia. Since the specific receptors are probably tactile, this disturbance of the pleasurable character of the sensation must be due to local interference with irradiation or summation.

The painful and pleasurable sensations thus form a group unified by peculiarities of summation and irradiation which are not exhibited by any other sense modalities. No other modalities show the pathological alterations in 'affective' character which occur in this group. The conclusion seems justified that the pathological changes in 'affect' are bound up with the special properties of conduction of this limited group of somesthetic impulses. These properties are not specific to the thalamus, but occur wherever the impulses reach a center, spinal gray, medulla, or thalamus. The especial importance of the thalamus may be ascribed to the fact that it contains the largest and most intricate nuclei within which these impulses are relayed and thus offers maximal opportunity for any abnormalities of conduction.

We do not yet know enough about the behavior of somesthetic impulses in summation and irradiation to understand their various central effects, but can infer something from the properties of the motoneuron pool (Sherrington, 29). The suggestion has been made that when a pool has alternative outlets, the first impulses to arrive may prime one path, which will then be facilitated by impulses which might otherwise have initiated another reaction. Under pathological conditions not only primacy, but also number and rate of succession of impulses might be determining factors, and a slight change in timing or intensity could alter the path of afferent discharge and so the subjective character of the effects of the stimulus. Changes in relative dominance among impulses from various receptors would account for alterations in 'affect' resulting from partial destruction of conduction paths as well as from injury to centers and so explain hyperalgesia from injury to peripheral nerve and to spinal tracts.

This is, in a way, a theory of release from inhibition (change in dominance of impulses in a center) but it does not involve, as does Head's theory, the conceptions of the specific inhibition of a center, of a localized source of inhibitory impulses, or of a restricted center for the emotional reinforcement of sensory impulses.

The available evidence justifies the conclusion that the 'affective' character of somatic sensations is correlated with their peculiarities of conduction, and probably their relations to vasomotor reflexes, and that their 'affective' character may be altered by lesions which disturb conduction at any of the synaptic junctions through which the impulses are relayed. There is no support for the assumption that the thalamus has a unique influence upon the 'affective' character of the sensations.

Pleasure-pain and affect.—One further question, concerning the pathological changes in somesthetic sensation produced by lesions in the afferent paths must be discussed. Are they primarily changes in affect, or changes in the intensity and localization of specific sensations? The relation of pain and sensations of pleasure to unpleasantness and pleasantness is still a controversial matter. There seems however to be a general agreement that the sensations are correlated but not identical with affect. Tickle may be unendurable, the warm flush of fever decidedly unpleasant. Pain may be sought as a source of masochistic pleasure. Investigators who still argue for a sensory basis of affect (Nafe, 25; Hoisington, 20) do not identify it with pleasure and pain, but with bright and dull pressures. It cannot be argued, therefore, that the sensations of warmth and tickle constitute pleasantness and of pain unpleasantness. They may induce, but are not themselves, affect.

The reports of the patients with hyperalgesia are concerned primarily with changes in the character of sensation; the pain is more intense, more burning, more diffuse on the affected than on the normal side. The reports concerning the changes in sensations of warmth may be similarly interpreted. The abnormality is primarily in the nature of the cutaneous sensations. They are intensified, rendered more diffuse and persistent. Such pathological sensations are no more to be identified with affect than are normal ones. Their association with nervous lesions does not provide evidence for a specific affective center, but only shows that the affective reaction, whatever be its nature, is more intense for sensations of pathological quality or intensity.

We may question, then, whether the reported observations on clinical cases with thalamic lesions are in any way relevant to the question of the existence of an affective center in the thalamus. Not only are there no general changes in affect produced by such lesions, but even for the limited group of somesthetic sensations we cannot be sure that the pathological disturbances are affective and not merely sensory.

THE MAINTENANCE OF EMOTIONAL ACTIVITY

One of the most important psychological functions which has been ascribed to emotion is the maintenance of attitudes or of motivational tension. Current theories of psychopathology are based upon 'unconscious emotional drives' and, in fact, the whole development of modern dynamic psychology centers around such concepts. Perhaps there is no need to assume an extraneous 'drive' to account for perseveration of behavior or of hysterical symptoms, but the persistence of activity in the absence of environmental stimuli is one of the major problems of physiological psychology and is characteristic of much behavior called emotional. Does the thalamus provide a mechanism for this dynamic function?

Theories proposed to account for persistent behavior have assumed the maintenance of activity, either by circular reflexes (Kempf, 21), by persistent endocrine stimulation of muscles with resultant sensory excitation, or by some sort of reverberation of tonic excitation within the central nervous system (Ebbecke, 13). Presumably, if the dynamic aspects of emotion derive from the thalamus, the nucleus there must maintain excitation, either by initiating circular reflexes, or by its own internal activity. Lacking direct evidence for such activity, we can only inquire whether the release of the thalamic emotional center from cortical inhibition increases the duration as well as the excitability of emotional response.

The persistence of emotional expression.—Bard (1, 2) has reported that there is practically no after-discharge in the sham rage of the decorticate animal. The reaction continues so long as the irritating stimulus is applied, but stops immediately with the termination of the stimulus. From his

descriptions fear and sexual reactions seem to persist somewhat beyond the stimulus, but not apparently longer than in normal animals. Unequivocal data are not available concerning the duration of emotional disturbance in normal cats. In the rat, with which I am more familiar, the after effects of a fight may persist and render the animal unsafe to handle for several hours, and it is probable that similar persistence of emotional disturbance can be demonstrated in the normal cat. There is apparently no indication of continued emotional reactions in decorticate animals which corresponds to the persistent emotional upset which is so often seen in normals. There is thus no evidence that the thalamus serves as a reservoir of emotional tension or contributes in any way to the motivational aspects of emotional behavior.

Restlessness.—Restless pacing is a characteristic of many decorticate animals. Although it might be taken as evidence for some subcortical driving mechanism, it can also be explained in terms of the general increase in excitability of lower centers after decortication and in itself does not constitute evidence for a specific dynamic center in the thalamus, any more than do the contractures of hemiplegia, or the stepping movements of a low spinal preparation. This applies also to the excitement and restlessness in cases of frontal lobe injury. Some tendency to maintain activity once initiated may be considered as characteristic of all central nervous function (Brown, 8). There is certainly no evidence that it is more characteristic of the thalamus than of other regions.

SUMMARY

Among the variety of phenomena which have been included under the topic of emotion are (1) a supposedly unique experience, (2) the hypothetical impulses and drives which make man the neurotic animal, and (3) such bodily activities as are not directly orienting, locomotor, manipulative, digestive, or linguistic. A review of the evidence fails to reveal participation of the thalamus in any but the third of these classes. The thalamus contains centers in which some, at least, of the patterns of expressive movement are integrated.

These, however, must be regarded as strictly motor centers, since the evidence for dissociation of expression from emotion, which has been advanced against the James-Lange theory is equally applicable to show that the thalamic centers for expression cannot contribute the quality of emotion.

The supposed evidence that the thalamus adds the affective or emotional character to sensations breaks down completely when subjected to critical analysis. The affective changes resulting from thalamic lesion are restricted to a small group of somesthetic sensations and cannot be interpreted as a general change in affectivity. The changes correlate definitely with the special properties of conduction, summation, and irradiation of this group of sensory processes and not at all with a specific locus in the thalamus. The pathological changes following thalamic lesions are primarily in the character of the sensations, in intensity, duration, localization, and are therefore not relevant to the problem of affect. There is no evidence whatever that the thalamus contributes facilitative impulses which might form a basis for the motivational aspects of emotion. Thus, the only part of the thalamic theory of emotion which has factual support is the localization of motor centers for emotional expression within the hypothalamus. It seems certain that these motor centers do not contribute directly to other aspects of emotion and there is no evidence for the existence of other affective or emotional centers.

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PREPARATORY SET (EXPECTANCY)—A DETERMINANT IN MOTIVATION AND LEARNING

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I

In an experiment recently conducted by the writer, an attempt was made to determine whether human beings learn to make a galvanic skin response to a flash of light of five seconds' duration more readily and more consistently (a) when the light is invariably followed by a brief electric shock (of 200 milliseconds duration) or (b) when the light is followed by shock only if a response does not occur within the five-second interval during which the light is on. Without using precisely this terminology, Freud (15, 16) has long emphasized the importance of conditioned fear reactions, in both normal and abnormal behavior, as signals which warn individuals against danger situations. Assuming that the G. S. R. is a reliable indicator of such a reaction, it seemed to follow that, by the law of effect, a conditioned fear (galvanic skin) reaction which was 'rewarded,' *i.e.*, was not followed by the shock, would be more strongly reinforced than a conditioned fear reaction which was not so 'rewarded,' *i.e.*, was invariably followed by shock. In other words, in the experiment referred to, the law of effect would seem to demand that learning proceed more rapidly in situation (b) than in situation (a). On the other hand, since the light was *more frequently* followed by (associated with) the shock in situation (a) than in situation (b), the association (conditioned response) theory would require more rapid learning in situation (a). It

¹The writer is indebted to Mr. Douglas G. Ellson and to Mr. Glenn L. Heathers for many stimulating suggestions and criticisms concerning the contents of this study. The experiment reported in the first section was to have been carried out jointly with Dr. J. M. Porter had not unanticipated circumstances made collaboration impossible.

thus seemed that this experiment might offer an exceptionally favorable opportunity to compare the relative validity of the two major theories of the learning process, since the results would presumably be uncomplicated by 'voluntary' factors, as they would have been if a striped-muscle response had been used (*cf.* Schlosberg, 48, and Hunter, 27).

However, it soon became evident that it was useless to carry out the experiment as originally planned, for it was noted during the early stages of the study that the subjects almost always showed a sizable galvanic response to the light on its *first* presentation, before the shock had ever been presented. Ordinarily such a reaction would merely be referred to as the "unconditioned response to the to-be-conditioned stimulus"; but the inaptness of this expression was soon demonstrated by the observation that the G. S. R. to the first presentation of the light was either quite small or entirely absent *if the electrodes through which the shock was to be administered to the subject were left unattached*. When, on the other hand, the electrodes were then attached to the subject, a vigorous 'unconditioned' response made its appearance as soon as the light was again presented.

This response to the light would, of course, eventually extinguish, but it was found that extinction could be prevented quite as effectively when the light was only occasionally followed by the shock [condition (b)] as when it was invariably followed by the shock [condition (a)]. Thus, in keeping with the results of numerous investigators who have studied the influence of 'knowledge of results' upon learning (6), of Dunlap (8) in his work on 'negative practice,' of Skinner (54) on experimental extinction, and of Thorndike (57) in a variety of experiments reported in his 'Fundamentals of Learning,' this finding of the present investigation would seem to point to the conclusion that the mere frequency with which a given response is elicited by an unconditioned stimulus, paired with an originally neutral (secondary) stimulus, is not related in any very direct or important way to the establishment or persistence of new stimulus-response relationships.

But it also soon became clear that the results of the in-

vestigation just described could likewise not be said to support the law of effect. On several occasions the writer removed the electrodes from subjects who had already received several shocks in combination with the light and then tested the effect of the light alone. Regardless of whether the subjects had been shocked under condition (a) or condition (b), the removal of the electrodes was regularly followed by a prompt and virtually complete disappearance of the response to the light; but with the replacement of the electrodes, the response to the light returned with undiminished vigor, without further administration of the shock (*cf.* Porter's investigation of the conditioned eyelid reaction, 41).²

These findings thus aroused the suspicion that not only the 'unconditioned' response to the light but also the 'conditioned' response which could be elicited after the light had actually been paired with the shock were largely dependent upon the subject's *state of expectancy or preparatory set, and did not reveal true learning*. But it could be objected, at least as regards the disappearance and reappearance of the 'conditioned' response, that the presence of the electrodes on the subjects had become an essential part of the total stimulus pattern and that one would not necessarily expect to obtain this response when the stimulus pattern was altered by the withdrawal of this component. The unsoundness of this criticism was, however, easily demonstrated by substituting for the actual removal of the electrodes merely a system of buzzer signals which indicated to the subject when the light might be followed by shock and when it definitely would not be. The photographic records reproduced in Fig. 1 show that even though the *external* stimulus pattern is thus held rigidly constant, the 'conditioned' G. S. R. to the light could be either suddenly 'extinguished' or suddenly 'disinhibited,'

² Since the present study went to press an article by S. W. Cook and R. E. Harris (2), entitled 'The Verbal Conditioning of the Galvanic Skin Reflex,' has appeared in which the authors report results which are in complete agreement with the findings just described. During recent months a number of studies have also been published concerning the rôle of so-called 'verbal factors' in the conditioning of certain other responses as well.

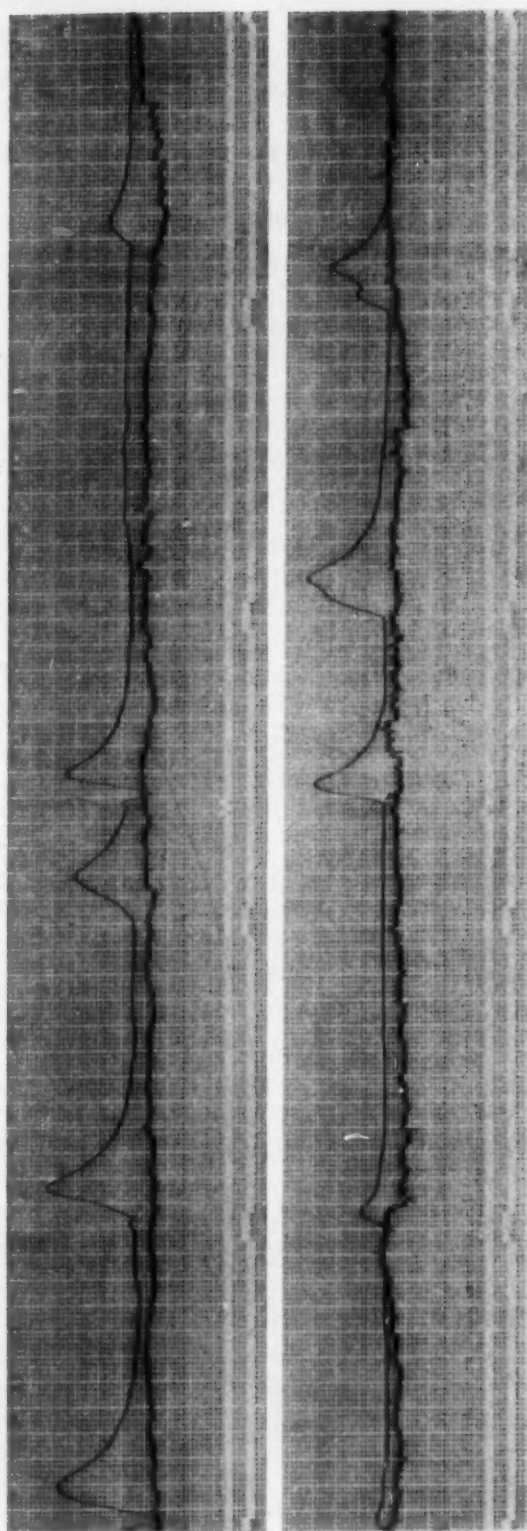


FIG. 1. The top (black) line in the accompanying photographic records shows the galvanic skin reactions made to a weak flash of light (indicated by the elevations in the lower signal line) under two sets of conditions: (i) when the subject (left half of upper record and right half of lower record) believed that the light might be followed by an electric shock; and (ii) when the subject (right half of upper record and left half of lower record) was confident that the light would not be followed by a shock. Note the large 'conditioned' responses to the light when a shock was expected (and when the buzzer signal—upper signal line, indicating change from one set of conditions to the other—was sounded) but the virtually complete absence of response to the light when the shock was not expected. The upper and lower records are continuous and were cut only for purposes of reproduction. The uneven black line just below the G. S. R. line shows the pressure with which the subject held a pneumatic bulb in one hand; no significant changes in muscle-tonus are to be observed. The heavier vertical white lines mark five-second intervals. Actual shock (which would have been indicated by a reflex contraction of the hand holding the pneumatic bulb) was presented at no time during the course of the record.

merely by changing the subject's internal state of readiness.³

II

A number of experimenters, notably Liddell, James, and Anderson (30), have observed with animal subjects that a conditioned response which has been subjected to experimental extinction or which has disappeared under certain other conditions (distraction, lapse of time, drowsiness, or the like) can often be resuscitated by one or two presentations of the unconditioned stimulus, without the accompanying presentation of the conditioned stimulus. Here the gain in excitatory strength of the conditioned stimulus can obviously not be due to pairing with the unconditioned stimulus. Generalization, or irradiation, of conditioning, in which stimuli which have *never* been paired with the unconditioned stimulus acquire new excitatory tendencies, is, of course, well recognized. Schlosberg (48), working with rats, has shown, as an especially dramatic illustration of this phenomenon, that a conditioned leg retraction which has been established by paired presentation of a buzzer and a shock can subsequently be elicited also by a flash of light. Pavlov (39) has attempted to explain generalization of conditioning by positing the spreading of a hypothetical excitatory brain state. A much simpler explanation, and one which would seem to be more in line with established physiological principles, is that generalization is a function of the degree of readiness or preparedness of a given reaction system and that by virtue of the development of an unusually high condition of readiness, stimuli which would ordinarily be without visible effect are now capable of eliciting the response for which the pre-existing set was appropriate.

If generalization of conditioning is thus conceived as due to an augmentation of readiness on the part of a given reaction

³ It is, of course, essential that the subject have complete 'confidence' in the experimenter and definitely 'believe' the buzzer signals which are presented to him. That conditioned galvanic responses *can* be set up, by using more severe shock, which do not disappear as soon as the danger of further shock is withdrawn is not disputed (cf. Miller, 34). Such conditioning presumably implies true learning, rather than mere changes in preparedness, and will be referred to again in later sections.

system, one is prompted to ask to what extent the responses which occur to the stimulus which *has* been paired with the unconditioned stimulus may also be dependent upon this factor of preparedness. Schlosberg (45), Miller and Cole (33), and others have shown that a conditioned response can be more easily established if the *general* level of psycho-motor tension is increased by the concomitant execution of some voluntary activity, such as clenching the fists (the facilitation of simple reflexes by this method being, of course, well known). That a specific preparatory set or attitude of expectancy makes the response for which the preparedness is appropriate liable to being set off, or 'tripped,' by inappropriate stimuli is common knowledge. When waiting in a state of tense preparedness to execute a particular action, everyone has had the experience of having this response prematurely initiated by some accidental noise or other form of stimulation which would ordinarily have no tendency to elicit such behavior. This seems especially likely to occur in 'nervous' individuals; Schlosberg (45, 46) has specifically commented upon the fact that 'nervous' individuals also make the best subjects in at least some types of conditioning experiments. (For a more general discussion of the psychological significance of preparatory sets and tensions, see Dashiell (5) and Freeman (12).)

Another suggestive parallel is that between experimental conditioning and the inclination of subjects in simple reaction-time experiments to make anticipatory, or 'false,' responses; in the one case the response is 'tripped' by the so-called conditioned stimulus, in the other case by some accidental stimulus. That readiness or preparatory set is induced in conditioning experiments by what may be called a coercive stimulus and in the reaction-time experiments by verbal instructions (plus the 'ready' signal) does not seem a sufficient reason for making a categorical distinction between the new stimulus-response relationships which are likely to appear in the two situations. The probable identity of the so-called false reactions and at least certain forms of conditioned responses has recently been emphasized in a series of experiments by Rexroad (43, 44), who states: "In these studies the

buzzer signal becomes a signal for the light and induces a readiness to respond to the light. When this readiness is highly developed, any stimulus will trip it into overtness" (44, p. 475).⁴

Hull (25) has suggested a division of conditioned responses on the basis of whether they have the short-latency of a reflex or the longer latency of a voluntary reaction. The development of the former type of response, which he terms *Alpha* conditioning, he regards as due merely to a more or less temporary sensitization or augmentation 'of the original unconditioned reaction to the conditioned stimulus'; the development of the latter, which he terms *Beta* conditioning, he seems to identify with learning proper. Inasmuch as it would now appear that long- as well as short-latency conditioned responses may be produced through 'sensitization,' a more significant criterion for differentiation would be between (i) new stimulus-response relationships which can be demonstrated only when there is a pre-existent readiness to make the particular response and (ii) new stimulus-response relationships which can be demonstrated in the absence of a pre-existent readiness to make the response.⁵ Only in the latter situation can learning be unambiguously inferred to have occurred.

It seems to be generally assumed that 'external inhibition,' 'extinctive inhibition,' 'generalization,' and various other effects which have been discovered in conditioning experiments relate to the learning process. In view of the widespread failure to discriminate between instances of real and only apparent learning, the significance of these subsidiary principles remains uncertain; they may turn out to be due merely to distraction, competition of rival reaction systems, and other familiar phenomena (cf. Dunlap, 9; Razran, 42; and Wendt, 62). Max Meyer has, in fact, long emphasized the difference between changes in stimulus-response relationships which are due on the one hand to what he has called "pre-

⁴ See also Dodge (5).

⁵ It seems almost certain that learning may also take the form of new connections between a given stimulus and a preparatory set to make a given response; but this type of learning has been relatively little investigated to date (cf. Rexroad, 44). See footnote 10.

occupation" and, on the other hand, to "true learning." The former "can generally and by proper methods be caused to be completely gone in a few minutes" and "is something entirely different from habit, at least in a normal being . . ." (32, p. 125). Meyer makes it clear that by "preoccupation" he means what other writers have called "readiness for business" and "attention," but he refuses to use this last term because of its "faculty" implications. The term "immediate memory," as opposed to "remote memory" (true learning), has also been used by other writers with much the same connotation. The distinction which the present writer is here pointing out is therefore by no means new, but it has for some reason been almost completely ignored in the interpretation of the experimental work on learning which has been done by conditioned-response methods. That confusions and misapprehensions have as a result arisen seems inescapable and points the need for critical re-examination of the existing facts and theories in this entire field.

III

In the preceding sections it was shown how the phenomenon of preparatory set or expectancy may be responsible for the appearance, independently of learning, of new stimulus-response relationships which are often mistakenly assumed to be due to learning. In the following sections an attempt will be made to indicate how preparatory set may also be involved in true learning and how, by positing such a relationship, certain advances can be made toward a more unified theory of the learning process.

In the early formulations of his theory of learning Thorndike (56) posited two major principles: the so-called law of exercise and the law of effect. The former was essentially a restatement of the traditional law of association through temporal contiguity. The law of effect, on the other hand, was an outgrowth of hedonistic thought and was assumed to imply two sub-principles: (i) that stimulus-response connections or relationships are strengthened (stamped in) when followed by pleasurable or satisfying consequences, and (ii)

that stimulus-response connections are weakened (stamped out) when followed by painful or annoying consequences.

Subsequent writers who have been primarily concerned with the explanation of trial-and-error learning, that is to say, learning in which goal-seeking is conspicuous, have tended to regard the law of effect as adequate for their purposes and have minimized or ignored Thorndike's law of exercise. On the other hand, writers who have been primarily concerned with the explanation of learning in which goal-seeking is less obvious, as in many forms of conditioning for example, have relied principally upon the law of exercise. For a time investigators in the field of learning became more and more sharply distinguishable according to whether they emphasized the law of exercise or the law of effect. There are, however, certain facts which prevented either type of theory as traditionally formulated from being entirely satisfactory. There is, for example, a large body of evidence (previously referred to in Section II) which indicates that under certain conditions, frequency of temporally contiguous associations seems to have no significance as far as learning is concerned. On the basis of this evidence, much of which he has himself amassed, Thorndike (57) has recently repudiated the law of exercise. Many experimenters in the field of conditioned responses continue, on the other hand, to find the law of effect unacceptable as an explanation of certain forms of learning with which they deal. According to Thorndike's original statements, a painful or annoying state of affairs was supposed to weaken stimulus-response connections. The observed fact is that by pairing a formerly neutral stimulus with a recurrently presented painful (noxious) stimulus, the formerly neutral stimulus tends to acquire the capacity to elicit a response which was formerly elicited only by the painful stimulus. In the case of these so-called conditioned defence reactions, the occurrence of a painful state of affairs appears not only to strengthen but actually to establish this new stimulus-response relationship, instead of weakening it. In the light of this type of observation, many investigators reject the law of effect and adhere to the law of exercise or some variation of it, despite the evidence which can be mustered against it.

Schlosberg (49) has just published a study in which he recognizes the foregoing problem and attempts to resolve it by assuming that there are really two types of learning process, the one in which the law of effect is operative and another in which the law of exercise predominates. This, in the opinion of the writer, represents a premature abandonment of the goal of a more unified and parsimonious view of the phenomena of learning (*cf.* Skinner, 55, and Hilgard, 19).

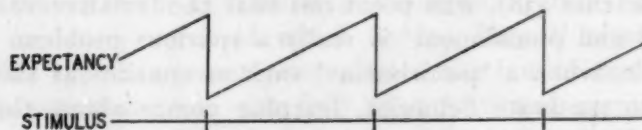
An important step in this direction has been taken by Thorndike (57). He advances experimental results which he interprets as indicating that the pronouncement of the word 'right' tends to strengthen stimulus-response connections but that the word 'wrong' has no weakening effect. On the basis of this and related evidence, Thorndike has been led to question the validity of his previous assumption that a painful state of affairs weakens connections and to suggest that connections are influenced only by satisfying or pleasurable states, and this always in a positive direction. This type of analysis has been extended somewhat further by Muenzinger and Fletcher (38), who point out that the 'relative value of reward and punishment' is really a spurious problem. For example, when a 'punishment' such as continuous shock is used to motivate behavior, learning comes about through *escape* from shock; just as when hunger is used as a motive, learning is likewise due to escape from hunger (eating).⁶ In both cases, according to these writers, the *reduction* of stimulus tension is the important factor; increase in stimulus tension (punishment or deprivation) is important to learning only in so far as it eventually makes possible a more drastic and effective tension reduction. Since reduction in tension is generally rewarded as satisfying and an increase in tension as annoying or painful (see Section VII), this analysis, therefore, neatly agrees with Thorndike's revised view that the operation of the law of effect and learning depend exclusively upon satisfying states of affairs.

IV

The analysis just presented falls short, in the opinion of the writer, of making the law of effect acceptable as a universal

⁶ Cf. Hollingworth (20), Holt (22), Guthrie (18), and Young (66).

theory of learning in one important respect: it does not adequately account for the fact that a *momentary* painful stimulus can, as pointed out above, indisputably be used to produce learning; here *escape* from stimulation of such brief duration can scarcely be regarded as meaningful. There is, however, a promising way of obviating this difficulty. In experiments with human subjects in which a momentary painful stimulus is recurrently presented, the subjects commonly report that they begin to experience shortly after each presentation of the stimulus a mounting feeling of inner tension which they variously describe as 'anticipation,' 'dread,' 'apprehension,' 'expectancy,' 'anxiety,' etc. This subjective experience is reported as rising toward a maximum and then dropping suddenly with the actual occurrence of the noxious stimulus. Although individual differences, frequency and intensity of the stimulus, number of times it has previously been presented, etc., are undoubtedly influential here, this subjective, cyclic phenomenon can be represented in relation to the recurrent noxious stimulus roughly as follows:



Wever (63), working with cats, Upton (61) with guinea pigs, and Schlosberg (47) with rats have all reported objective evidence of the cyclic phenomenon just described. Curtis (3, p. 3) has recently given an especially clear description of the phenomenon as it appears in the pig. This writer reports that, following the presentation of a recurrent shock to the pig's front leg, there is a period "of several minutes of abnormal quiet, as shown by the regular breathing and smoothness of activity. Suddenly respiration becomes irregular, the pig begins to growl, crouch, brace the body, and the performance culminates in this case with a squeal and sharp flexion of the leg in about ten seconds—the same length of time which the normal reaction requires. But in this case, neither signal nor shock was given by the experimenter. The whole thing

is what we have called with anthropomorphic enthusiasm a 'hallucinatory reaction,' although a better explanation probably is that the mounting tension which is to be released, trigger-fashion, by the anticipated shock signal, has grown so strong that the animal can no longer contain it. It is significant from this point that *the electric shock seems both in the pig and sheep to come as a relief, and is followed by a period of comparative relaxation before anticipatory behavior toward the next shock begins.*⁷

If, as the foregoing observations indicate, a recurrently presented noxious stimulus of brief duration is characteristically preceded by a period of heightened anticipatory tension⁸ and if this tension is markedly reduced immediately following the occurrence of such a stimulus, one can think of *escape from tension* as providing no less appropriate conditions for the operation of the law of effect than does escape from hunger, escape from protracted electric shock, or escape from any other motivating stimulation. And just as incidental, or secondary, stimuli which are temporally contiguous with those responses which are made at the time of escape from hunger, for example, become integrated with the hunger stimulus into a total stimulus pattern which, with repetition,⁹ becomes more and more specifically connected with these responses, so would it appear that stimuli which are temporally contiguous with

⁷ Italics added by the writer.

⁸ That expectancy or preparatory set may be modified through learning or even conditioned to formerly neutral stimuli is granted. But the assumption is here made that a preparatory set is not fundamentally a learned phenomenon; it is due rather to a basic tendency on the part of every reaction system, once activated, to show, following a relatively brief period of refractoriness, a more prolonged period of increased excitability. Experimental evidence in support of this assumption will be published shortly. See also footnotes 5 and 10.

⁹ In preceding sections evidence has been advanced in support of the contention that the mere frequency with which an incidental stimulus is associated with a given response, which was originally elicited by some other (primary) stimulus, has *no influence* upon the development of a functional connection between the incidental stimulus and this response. Repetition, or frequency of association, is important only when the response is followed by what Thorndike has recently called a 'confirming reaction' (satisfying state of affairs), *i.e.*, by the operation of the law of effect. Thus, contiguous association and frequency are both significant factors in the learning process but only insofar as they are conditions for the operation of the law of effect; in and of themselves they appear to have no importance to learning (*cf.* Hunter, 26).

those responses which occur at the time of escape from an anticipatory tension likewise become integrated with the anticipatory tension into a total stimulus pattern which, with repetition, becomes likewise more and more specifically connected with these responses. Eventually such incidental stimuli may acquire sufficient excitatory value as to be capable alone of eliciting the responses with which they have been temporally associated, without the accompanying presence of the original motivating stimuli (hunger, anticipatory tension, or the like).¹⁰ When this happens the evidence that learning has occurred is indisputable; but as long as an incidental, or secondary, stimulus is capable of eliciting a given reaction only when a preparatory set is already present for the execution of the response under investigation, the evidence as to whether learning has occurred is ambiguous. As pointed out earlier (Section I), in this latter situation the incidental stimulus may simply supply, through a process of stimulus summation, the straw which breaks the camel's back, independently of learning.

If anticipatory tension, or preparatory set, is thus acknowledged as capable of functioning as a drive or motive (as Woodworth, 65, has long insisted),¹¹ not only is it possible to account for the formation of conditioned defence reactions

¹⁰ They probably very quickly acquire the capacity to create a preparatory set, that is to say, an incomplete form of the response. This tendency for a formerly neutral (secondary) stimulus to acquire first of all the capacity to produce a preparatory set would, in fact, appear to offer an improved, more refined test of learning than is ordinarily available in conditioning experiments, where learning is usually not demonstrable until the secondary stimulus has been paired with the primary (unconditioned) stimulus so many times that the secondary stimulus has become capable of actually eliciting the given response in more or less complete form. The existence of a preparatory set, established by a secondary stimulus, could be tested for by comparing the size of the response made to the primary stimulus (a) when the secondary stimulus is present somewhat before the primary stimulus and (b) when the secondary stimulus is omitted. In other words, the extent to which the secondary stimulus proves capable of acting as an effective 'ready signal' could thus be used as an index of learning. An experimental attempt is shortly to be made to use this procedure as a test for learning; certain controls are of course necessary in this connection, but they can easily be provided. See footnote 5.

¹¹ Peak (40, p. 77) has likewise remarked that "the assumption that stimuli and sets are essentially different and may be distinguished . . . may prove to be founded on our lack of information."

on the basis of the law of effect and thus remove what would seem, in the writer's opinion, to be the final objection to the universality of the law of effect as a theory of the learning process;¹² it also thus becomes clear why in the case of human beings, learning can be produced without recourse to any of the common forms of motivation used with animals. If by instructions, supplied either by the experimenter or by the subject himself (in the form of an hypothesis), a definite preparatory set or state of expectancy is established, the occurrence of any reaction which is said by the experimenter, or thought by the subject to be 'right' is accompanied by a drop in tension (preparedness) and therefore, according to the law of effect, by learning. Symbolically induced sets can thus be conceived as sources of motivation which follow precisely the same laws as do hunger, pain, and the other so-called organic drives. Since symbols, or signals, may acquire the capacity (through prior learning) to establish preparatory sets which may then act as motives for further learning, it is a relatively simple matter to account for the development of so-called secondary or even more remote orders of conditioning. Ordinarily the preparatory sets or tensions which are established, at least in animals, by second- or third-order conditioned stimuli are so weak that they cannot be used to motivate still higher orders of conditioning. Finch and Culler (10), however, have recently shown that if an animal's general level of motivation or alertness to these stimuli is kept high by the occasional simultaneous presentation of a "general motivating or activating stimulus" (p. 600)—an electrical shock, administered to some indifferent part of the body—

¹² Holt (22) has stressed the factor of recency in learning, holding that of two or more responses elicited by a given stimulus situation, the *last* response made will gain in probability of occurrence upon subsequent presentation of the same stimulus situation more than will the response or responses which have preceded it. However, to speak of the 'last' response made to a given stimulus situation under the conditions stipulated by Holt is necessarily to imply that it is also the 'effective' (satisfying) response; for otherwise, if it were not effective (*i.e.*, did not substantially alter the stimulus situation eliciting it), the organism would presumably *continue* to react to the given stimulus situation. It seems to the present writer that, operationally, it makes little difference whether one assumes that the last, or effective, response is learned because of a hypothetical "openness" of the last used motor path" (Holt, 22, p. 95) or because of an equally hypothetical 'confirming reaction' (Thorndike).

the excitatory value of each successively higher order of conditioned stimulus can be kept great enough to be used to establish still higher orders of conditioning. These writers believe that by the use of "a *constant* energizing agent" (p. 597) of this kind, any desired level of higher-order conditioning can be attained and that such an agent "serves much the same purpose in the dog as do ordinary social incentives in everyday human learning" (p. 602).¹³

There is one other point which should perhaps be mentioned here. In experiments involving trial-and-error behavior, in fact, in most life situations, there is a fairly obvious cause-and-effect relationship between a particular response and the satisfying state of affairs (success) which follows. The cat in a problem-box pushes the 'right' latch or lever and gets food (escapes from hunger), and so on. But when a conditioned defence reaction is being set up to a recurrent electric shock, why, as reported above, is the occurrence of the shock followed by a drop in the existing anticipatory tension? Eschewing a number of possible anthropomorphic explanations, the writer suggests that tension reduction occurs under these conditions precisely for the reason that the shock elicits those reactions for which the pre-existing tension or preparatory set is specifically appropriate. In another connection the writer (37) has observed in rats which were being subjected to a gradually increasing intensity of shock that the occurrence of a response, even a fairly small one, seemed to have a temporarily depressing effect upon the motivational value of the shock, at least at low intensities. In other words, there seemed to be, with the slow mounting of the shock, a concomitant mounting of inner tension (evidenced by increased rigidity of posture, disturbances of breathing, etc.) which, however, was temporarily dissipated by actual bodily

¹³ Tolman has repeatedly emphasized the importance of 'sign-gestalt-expectations' or 'hypotheses' as determinants in the learning process. Recently he has indicated that by these expressions he really means preparatory sets. However, instead of using the concept of preparatory set as a means of attempting to formulate a more unified theory of the learning process, he has been led to posit, not two, but "some seven different kinds of learning corresponding to some seven distinguishable classes of . . . environmental sequences" (59, p. 203). The present writer is unable to follow Professor Tolman's reasoning.

movement. It is conjectured that the occurrence of the response for which a preparatory set is specifically appropriate (and perhaps even other responses) normally reduces the tensions of which this preparatory set is composed.¹⁴

V

Although psychological interest in the past has centered mainly upon the problem of the acquisition of new behavior and only incidentally upon the elimination of old behavior, a comprehensive theory of the learning process must account for both phenomena. The law of effect, as originally formulated, did provide an explanation, not only of the formation of new-stimulus response connection, but also of the weakening or suppression of pre-existing connections. But as already pointed out, this latter aspect of the law of effect has now been, for good reasons, repudiated. If, however, the law of effect, in its positive form alone, is to be seriously considered as a universal theory of learning, how can the obvious fact be explained that established forms of behavior can be eliminated? Modern emphasis upon problems of 're-education,' especially in connection with work in the field of delinquency and the psychoneuroses, gives this question especial importance. While still acknowledged as a probable psychological reality, forgetting, conceived as the passive decay (due to the mere passage of time) of infrequently exercised responses, is today attracting much less interest as a psychological problem than it formerly did. The very fact that true forgetting, by definition, occurs only in infrequently used reaction systems

¹⁴ When the anticipatory tensions are excessively intense, *i.e.*, when they are *not* significantly diminished by the reactions which they help to produce, one observes, not learning, but the ultimate demoralization of behavior and frequently some form of 'nervous breakdown.'

Another point which cannot be fully discussed here is that defence reactions may sometimes apparently become conditioned in *one presentation* of the appropriate combination of stimuli. It follows from the above line of argument that a genuine conditioned defence reaction can be established only by *recurrent* elicitation in its unconditioned form. Because of the possibility of the *symbolic* repetition of events in man, it may well be that human beings do sometimes acquire conditioned defence reactions in what *appears* to be 'one trial.' An experimental attempt is now under way to determine whether or not this can also occur in lower animals, in which symbolic behavior is presumably negligible. Positive results would seem to constitute a serious threat to the validity of the above argument.

greatly reduces its significance. Behavior of a more vital character, concerned with the securing of food and the other necessities of life, with the maintenance of security, and so forth, has no opportunity of being forgotten in the strict sense (*cf.* Finch and Culler, 11); yet we know that such behavior does undergo radical and repeated changes. How does this come about?

Probably the most common reason for changes in the reactions of living organisms to their external environment are the waxing and waning of those internal stimulus situations which we refer to as organic drives. No one, for example, expects a dog when hungry to act exactly as he does when satiated. Here also must be mentioned, along with the so-called organic drives, those states of anticipatory tension, expectancy, or preparatory set discussed in the preceding section of this paper. A man who is living in constant dread of some impending (or fancied) disaster certainly behaves differently from what he does when free of such apprehensions. But all of these behavior changes are correlated with changes in the stimulus situation, external, internal, or both. The real question is, what is the process through which an organism which has previously responded to a given stimulus situation (internal and external) in a particular way becomes altered so that it later characteristically manifests a different type of reaction to essentially the same stimulus situation?

In Section III of this study evidence has been advanced for believing that stimulus-response connections are not weakened by punishment *per se*. The obvious inference seems to be that an established mode of behavior disappears only when, as a result of failure on the part of this behavior to meet with the accustomed satisfactions, the organism goes on to new (at first often random) behavior which eventually may bring the sought-for effects. Thus, the early reaction pattern is eliminated, but only in the sense that it is superseded, or overlaid, by a new form of behavior.

Wendt (62, p. 280) has presented a cogent argument in support of this thesis, which he has expressed by saying that "The loss of one mode of response is accompanied by a shift

to another mode of response. . . . That is to say, an activity is inhibited, when some other reaction system takes its place." Wendt's study is intended primarily as a repudiation of the Pavlovian concept of inhibition as a hypothetical brain state or substance which is generated by the unsuccessful (non-reinforced) repetition of conditioned responses and which reacts back upon the neural pathways involved in these responses so as to dampen or suppress the behavior in progress. Aside from the fact that this hypothesis is logically circular, it is not suited to account for the more or less *enduring* changes which manifestly do occur in behavior. The assumption that learning is superseded only by new learning (*cf.* Guthrie, 18) seems to be the only available alternative.¹⁵

Freud (14, 17) has given extensive consideration, in a somewhat different context, to this problem, and as a result has evolved the concepts of repression and regression. According to Freud, habit 'progression' occurs only when conflict, or 'ambivalence' (produced by failure of accustomed satisfaction, or deprivation, or by active punishment) arises. If the ensuing struggle between the old and new habit systems proves to be intense and heavily laden with painful emotions, the resolution of the conflict is often accomplished through one of the competing reaction systems being completely 'driven from consciousness,' *i.e.*, repressed (*cf.* Sears, 51). But this is not to say that the vanquished habit, or reaction tendency, has been annihilated. On the contrary, it is supposed to remain intact, merely dissociated from consciousness and the main stream of behavior. In fact, repressed reaction tendencies are assumed by Freud to be uniquely protected from the modifying influences of other conscious process by the very fact of their repression ('lack of contact with reality'). By weakening the 'forces of repression' (*i.e.*, the competing reaction systems) through the techniques of psychoanalytic therapy, reaction systems which have apparently lain dormant for years as far as either the subject's behavior or consciousness is concerned (but which may have

¹⁵ It seems probable that certain forms of innate, reflexive behavior can be modified through habituation, often to the point of virtual elimination and for relatively long periods, by a process or processes very different from learning (35).

come to produce 'symptoms'), often re-assert themselves in much their original form and vigor, thus supporting the hypothesis that reaction systems which are superseded by competing reaction systems are not necessarily destroyed in the process.

Another confirmation of this thesis is the psychoanalytically observed phenomenon of regression. Here an earlier (often infantile) reaction system which has remained submerged, or repressed, perhaps for years, may often, because of the weakening (through various processes) of the reaction system which superseded it, suddenly emerge and begin to manifest itself anew. The present writer (37) has recently been able to produce in white rats what appears to be at least an experimental analogue of regression as observed clinically. In the investigation cited, habit (*A*) was established in one group of animals and was then superseded by another (more adequate) habit (*B*). However, when the execution of habit (*B*) was made moderately difficult, the animals in this group promptly reverted, or 'regressed,' to their earlier habit (*A*), thus showing that it was still functionally intact. Animals which were trained only on habit (*B*), on the other hand, continued to execute this habit after the introduction of the obstacle; that is to say, they did not 'regress.'

Thus, the foregoing observations and numerous other considerations which might be mentioned consistently point to the conclusion that learned behavior is ordinarily changed only by the learning of new behavior, which supersedes and suppresses (or represses) but does not destroy the earlier reaction tendencies. In Thorndike's (57, p. 277) words, "rewards in general tend to maintain and strengthen any connection which leads to them. . . . [Punishments] weaken the connection which produced them, when they do weaken it, by strengthening some other connection." Such a conclusion obviates the necessity of having any special principle, such as originally contained in the law of effect, to account for negative learning, as opposed to positive learning.

VI

Attention has already been called (Section IV) to the tendency of highly developed preparatory sets to go over into overt action, before the actual occurrence of the expected, or prepared-for, stimulus; 'coming forward' is a common characteristic of certain types of conditioning and again points to the probable connection between this phenomenon and set (*cf.* Spragg, 53). In general, observations in this field have been restricted primarily to anticipatory, or false, *reactions*, which are easily observed and recorded, but the same principles seem also to be operative in connection with perception. In the quotation previously cited, Curtis has suggested that the anticipatory reactions made by his experimental animals might be interpreted as due to an 'hallucinatory' perception of the shock stimulus. This was frankly only a surmise which could not be directly confirmed. However, in an experiment with human subjects, undertaken for quite a different purpose, the writer has recently obtained evidence which clearly support Curtis' conjecture.

In the investigation referred to, the subjects were instructed, as part of a more elaborate procedure, to press down a key with the index finger as soon as they felt a repeatedly presented electrical shock which started at zero intensity and gradually built up. In the preliminary stages of this study, it happened that apparatus difficulties occasionally necessitated delays of two or three minutes between the time when the shock was turned off and the time when it could again be built up. During these delays the subjects would almost invariably push the key, indicating perception of shock when no shock whatever was present.¹⁶ Exploratory investigation has shown (1) that false responses of this kind occur only after an interval greater than that normally existing between successive presentations of actual shock; (2) that there seems to be a positive correlation between the promptness of the occurrence of the false response and the general

¹⁶ This finding would seem to have important implications for the response theory of sensation developed among others by Langfeld (28), but the scope of this paper does not permit their consideration at this time.

reactivity level of the subject; and (3) that the length of time which the false reaction persists shows individual variability. A photographic record of a typical false reaction of the kind here referred to is reproduced in Fig. 2.



FIG. 2. The ascending white lines in this record indicate a gradual building up from zero intensity of an electric shock applied to the forearm of a human subject. Subjects were instructed to push a key, connected with the signal marker (lower horizontal line), when the shock became perceptible and to hold it down until the shock was turned off. If, after a series of presentations of the gradually increasing shock, the shock was completely withheld for a time, subjects almost invariably pushed the key (see central region of record), indicating an hallucinatory perception of the shock. The heavier vertical white lines represent time intervals of five seconds.

By usually accepted criteria the false response shown in Fig. 2 is a pure exhibit of so-called temporal conditioning. As a result of repeated presentations of the shock (plus the instructions to depress the key as soon as the shock was felt), a recurrent preparatory set is established for the execution of this response. With the passage of sufficient time, this set becomes so strong that it goes over into overt behavior, in the total absence of the adequate stimulus. In all essentials, it is thus strictly parallel to a 'trace' conditioned response.

This type of false reaction can not only be regarded as an illustration of temporal conditioning, but it also provides proof that the subjects experience a concomitant sensory hallucination. Whipple (64) has reviewed a number of experiments in which the creation of an expectancy of the perception of warmth actually causes subjects to report this sensation. Brown (1) has found that comparable effects can be produced by suggesting to individuals that they are about to receive a faint electrical shock. And it has long been known that the task of determining the absolute threshold of virtually all forms of sensation is complicated by the tendency for subjects' judgment to be influenced by auto-suggestion.

The foregoing considerations indicate that expectancy is an important determinant, not only in what individuals do, but also in what they sense; the evidence here cited also provides an objective approach to the analysis of the nature of suggestibility (*cf.* Hull, 24, and Scott, 50).

VII

In the preceding sections of this paper the concept of preparatory set has been given to two-fold emphasis: on the one hand, as an explanation of a variety of behavior changes which are often mistakenly attributed to learning but which are really due to the mere 'tripping' of a pre-existing state of preparatory tension; and, on the other hand, as a motivational factor, by the postulation of which the law of effect, as recently reformulated by Thorndike, can be made more acceptable as the universal explanation of learning, applicable no less to the conditioning of defence reactions than to the formation of new habits through trial-and-error and other more obvious forms of goal-seeking behavior.¹⁷ A number of specific implications of this approach to the theory of learning have already been pointed out, but there remain a few related problems of broader scope which should also be at least briefly referred to.

The view that learning occurs only when a reaction-system which has been under tension undergoes tension-reduction has especial relevance for educational theory and practice. This way of regarding the problem of learning makes the adherents of formal disciplines and the more modern advocates of so-called progressive education methods in a sense both right and yet each only half right. The former stress the importance of diligent effort and high motivation, paying little attention, at least in theory, to the degree of

¹⁷ Some writers have objected to the law of effect on the grounds that it has to 'act backwards' in order to be effective; that is to say, the satisfying state of affairs, or 'confirming reaction,' comes *after* the stimulus-response sequence which it is supposed to reinforce or fixate. Backward conditioning, however, presents almost precisely the same problem, yet we now know—Pavlov's early dictum notwithstanding—that backward conditioning is an established fact (25). The fact that we are not as yet able to give a physiological, or chemical, or subatomic account of the law of effect likewise does not impair its validity or usefulness at the level of behavior (*cf.*, Troland, 60).

attendant success or satisfaction thereby attained; whereas the progressive educationalists emphasize the factor of success and tend to neglect the importance of pre-existing tensions and striving. Both of these factors are essential for efficient learning, and only confusion can result from the stressing of one to the exclusion of the other. The principle of alternating suspense and climax seems no less valid in education than in theatrical entertainment. A 'happy ending' can come only after a period of stress and strain.

It should also be observed that the use of preparatory set as a motivational concept seems necessary for an adequate analysis of various problems centered around human frustration; human frustration (and even animal frustration to some extent—*cf.* Tinklepaugh, 58) is determined not only by what the individual needs or is accustomed to, but also by what he is expecting or looking forward to. Some of life's keenest disappointments arise from the failure of expectations and hopes. May and Doob (31) have recently shown the desirability of using the concept of 'level of aspiration' as a motive in both individual and social behavior. This term borrowed from Hoppe (23), is directly related to 'task completion,' 'field closure,' and similar concepts which stress the importance of tension systems and their resolution in determining the development and form of behavior (*cf.* Lewin, 29). It seems not unlikely that by making more explicit and precise the concepts and hypotheses current in this field, an important step may be taken in effecting a rapprochement between certain of the so-called dynamic psychologies and the more traditional theories of behavior.

Shaffer (52) has recently shown the usefulness of the concept of psychological tension as an integrational device for bridging the gap between conventional psychology and the facts and theories of psychopathology. His views in this connection are, however, less consistent than those of Freud. The latter writer holds (13, p. 3) that, according to what he has termed the pleasure-principle, which he regards as the basic law of all mental life, "there is an attempt on the part of the psychic apparatus to keep the quantity of excitation

present as low as possible, or at least constant." This view, it will be seen, is entirely consistent with the revised version of the law of effect and the learning process discussed in the preceding pages. Shaffer, however, like Holt (22), introduces a complication in the form of the assumption that while much behavior is *abient* (i.e., functions so as to decrease the stimulation which produces it), other behavior is definitely *adient* (functions so as to increase the stimulation which produces it). The writer sees no necessity for such a dichotomy. It is true that living organisms sometimes go away from external objects and sometimes toward them; but in all cases the organism seems, in the final analysis, to be trying to escape from or lessen the motivational stimulation which has set it in motion.¹⁸ If the drive or motive, is for example, hunger, the organism has, to be sure, to go toward food before eating is possible; but, as Holt himself has pointed out (21, 22), the significant consideration here is not the approach to the food but rather the attempt to flee or escape from the hunger. Likewise, a man who is already fatigued almost to the point of exhaustion may continue to exert himself in order to reach a resting place; but certainly one would not say that his behavior, which at the moment may be motivated mainly by fatigue, occurs in order to *increase* this form of stimulation, even though it actually does have this immediate effect. The writer sees no fundamental objection to the generalization that in the final analysis behavior is normally always directed toward (persists until) the elimination of the stimulation which motivates it; if behavior sometimes does in fact increase certain types of stimulation, this is only incidental to the organism's progress toward the elimination of the drive stimulus.¹⁹

Holt uses the concept of adience primarily in an attempt to show that those reflexive and instinctive responses which are ordinarily regarded as inherited are in reality learned (see

¹⁸ If the proverbial moth sometimes flies toward or even into the flame, the flame can be said only to have reflexly *directed* the behavior; it did not motivate it. The moth *flies* because it is hungry, cold, sexually excited, or the like.

¹⁹ A number of other writers have weakened their discussion of the problem of motivation by introducing dichotomies, such as Troland's (60) 'beneception' and 'nociception' and Tolman's (59) 'positive demands' and 'negative demands.'

Mowrer, 36). Although Holt presents an ingenious hypothesis as to how some such responses, *e.g.*, grasping, may be individually acquired, the present writer is unable to see how 'tumbling' and 'rolling' in certain strains of domestic pigeons and many similar peculiarities of animal behavior which appear to follow along strictly Mendelian lines, can be regarded as anything but inherited. Since we are apparently forced to acknowledge the hereditary origin of at least some types of behavior, it seems a doubtful accomplishment to have succeeded in explaining a few other supposedly inherited responses on a non-hereditary basis, if in order to do this we have to introduce such a dubious concept as *adience*, one which in the writer's opinion confounds the whole learning problem.²⁰

It is true, of course, that children often object to going to bed even though they are obviously in need of rest, thus seemingly revealing an inclination to seek further stimulation instead of avoiding it. Adults, too, sometimes seek 'excitement' although already clearly over-stimulated and fatigued. But there is no real contradiction between these facts and the generalization that all motivated behavior is or tends to be avoidant (*abient*). Childhood, as we know, is characterized by conflicts and frustrations which are rarely exceeded or even approximated in later life. Like the adult who finds continual diversion and excitement necessary, the child often seeks to avoid the solitude of his room, where anxiety-laden phantasies well up and induce a state of greater psychological tension than ordinarily prevails at times when play and other distracting activities serve to keep internal conflict somewhat suppressed.

The objection may be urged that both children and adults often mutilate or injure themselves, thus seeming to violate

²⁰ Perhaps the grasping reflex which causes the young of certain primates to cling to the hair of the mother is inherited (if it is) precisely for the reason that this response would probably often not have time to be learned before, by virtue of its absence, the young animal fell from its arboreal home and was killed or abandoned; only infants with this reflex preformed would thus survive. A similar explanation can be advanced to account for nursing and many of the other so-called '*adient*' responses. Defence reflexes, such as the blink, pupillary constriction, and withdrawal flexions of the arms and legs, can, if necessary, be accounted for on the basis of learning through the law of effect (*cf.* Schlosberg, 49).

the pleasure-principle. Darbrowsky (4) has recently reported an interesting study on self-torture and comes to the conclusion that self-inflicted physical pain usually serves to alleviate a more excruciating form of inner, mental anguish. Freud (14) has repeatedly commented upon the tendency for hysteria and other neurotic symptoms to disappear whenever a form of physical suffering intervenes. Thus, masochistic behavior in general can be regarded as relieving, through the infliction of one form of pain or tension increase, another and less tolerable form of suffering.

It may be objected in this connection that that portion of the sexual act which precedes orgasm is characterized by increasing muscular activity, yet is definitely pleasurable. This criticism is based upon the mistaken assumption that psychological tension and muscular activity are positively and directly correlated. As suggested on an earlier page of this study, the relationship here is probably an inverse one: action presupposes the dissipation of tension (except fatigue tension). Thus as muscular activity increases in the sexual act, tension can be thought of as being more and more rapidly discharged. This process reaches its climax and terminates in orgasm.

It is true that human beings sometimes willfully deprive themselves of satisfactions which are immediately available in order to 'whet their appetite'—as in delaying a Thanksgiving dinner—so that gratification will ultimately be the keener. But this is not a real breach of the pleasure-principle, but instead only a complicated expression of it. A more serious problem is that offered by individuals who renounce certain 'worldly' pleasures in order, as they say, 'to lay up riches in Heaven.' Careful observation of such persons usually reveals, however, that these so-called renunciations are mere rationalizations used to gloss over the fact that, because of childhood training or social disapproval, they are actually *afraid* to enjoy the 'renounced' gratifications. Instead of recognizing this fact, it is, of course, easy to believe and say that one is simply being spiritually thrifty and saving up one's pleasures for a future life, problematical though it may be.

CONCLUSIONS

On the basis of the foregoing analysis of the problem of motivation and learning, the following conclusions, numbered to correspond to the sections from which they are drawn, seem warranted:

I. Apparent conditioned responses can be suddenly established and equally suddenly abolished in human beings (and probably also in animals) merely by controlling the subject's state of expectancy or preparatory set.

II. Evidence from a variety of conditioning experiments suggests that many of the subsidiary facts discovered in this field are due simply to changes in the nature and extent of the subject's preparedness or readiness to make the particular response under investigation and have no relation to learning proper.

III. An important step toward the elimination of objections to the law of effect as a universal theory of learning has resulted from the repudiation of that sub-principle which holds that stimulus-response connections are weakened by annoying or painful consequences.

IV. By acknowledging the mounting anticipatory tension (preparatory set) which precedes the presentation of a recurrent noxious stimulus as a form of motivation which is markedly reduced by the occurrence of the resulting response, it is possible to show that painful stimuli can be used to create and strengthen connections instead of weaken them.

V. A variety of considerations support the hypothesis that learned behavior is ordinarily changed *only* by the learning of new behavior, which merely supersedes and suppresses (or represses) but does not destroy the earlier reaction tendencies.

VI. The fact that anticipatory reactions, precipitated by an over-developed preparatory set, may be accompanied by an hallucinatory perception of the anticipated stimulus, points to a response theory of sensation and provides an approach to a stimulus-response analysis of suggestion.

VII. The foregoing propositions have a number of implications for education, psychopathology, and social psychology.

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TITCHENER ON MEANING

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Professor Higginson's description of Titchener's dilemma with the problem of meaning is very interesting and informative.¹ He makes two points: (1) that Titchener thought of meaning as being outside of psychology and was nevertheless at constant pains to establish the psychology of meaning, and (2) that Titchener was forced to recognize that meaning is, for the most part, "carried" unconsciously, although he believed that psychology is primarily concerned with consciousness. Certainly these points about Titchener's system ought to be emphasized, and Higginson does us the service of pointing them out once again.² On the other hand, I do not see why Higginson voices these observations as a complaint. Titchener took us a good part of the way from Wundt to the present, and why should he not write in 1915 as of 1915 with many of the constraints of 1900-1910 still hampering his thought? While it would be ungracious for me to quibble over various sentences of Dr. Higginson's, I wonder if I may not try to alter his evaluation of Titchener's contribution to psychology of meaning by setting down in five paragraphs a brief for the article that I should write on Higginson's subject. The reader of Higginson will see that I agree with him as to the facts about Titchener, although I should reverse his general evaluations.

1. Titchener first undertook to solve the problem of conscious meaning. He had from tradition via Wundt and Külpe the concept of existential process (sensations and

¹ G. D. Higginson, The place of meaning in psychology, *PSYCHOL. REV.*, 1937, 44, 491-504.

² Cf., e.g., E. G. Boring, *A History of Experimental Psychology*, 1929, 408, 411f., 428f., and esp. 588; *The Physical Dimensions of Consciousness*, 1933, 151f., 236f., and esp. 182. Of course it is no reproof to Dr. Higginson that I know my own books better than he knows them!

images), and the belief that psychology deals with mind ('dependent' experience) and not, except incidentally, with the subject-matter of physics. Thus he derived the context theory of meaning. It was not really a new theory; Berkeley had practically said it; but Titchener gave it explicit formulation and importance. In simple form it was this: it takes two mental processes to make a meaning. When a sensation or image is added ('accrues') to a sensation or image, one has a meaning in the form of a perception or an idea. It is no new thought to a logician that a meaning is a relation. Here we have Titchener saying that a conscious meaning is a conscious relation, and specifying the nature of the relation.

2. There is a paradox to theorizing. To have a theory of a thing is to get along without the thing. A physical (or behavioral, or operational) theory of consciousness substitutes for consciousness another formula, and there has been a great deal of confusion in recent systematic psychology as to whether such a theory of consciousness keeps consciousness in psychology or rules it out. Really what such a theory does is to ingest consciousness, bringing it so completely inside that it no longer seems to be what it was when it was outside. In the same way an atomic theory ingests solid matter. Does or does not physics deal with material objects, knowing them to be mostly electrons in motion? Titchener's theory of conscious meaning was similar. By reducing conscious meaning to relations of conscious contents, he could assert that he had got rid of meaning by having a theory of it. There is no puzzle here, if one realizes the validity of this paradox.

3. Titchener took from common sense the meanings for which he made a theory, but common sense was inconsistent. It said essentially that meaning is conscious, that meaning is knowledge, and also that knowledge is not always conscious. Thus Titchener began his theory in the tradition of psychology, considering conscious meanings. Then he noted that the organism 'knows' lots of things that it has no time to be conscious of—the habituated perceptions. He saw plainly that such perceptions are not conscious and he was

eager to promote progress by recognizing this fact. So he included in his theory the principle that the conscious context drops off from a perception under habituation and that the meaning is then carried unconsciously. For Titchener and many others of that date, *unconscious* meant *nervous*. Thus Titchener was led to say that the meaning is carried by a 'brain habit.'

4. In this way Titchener represents, in one of his most important theories, a stage in the transition from Wundtian mentalism to modern physicalism. Had operationism been the vogue even before 1915 (say, in 1908), Titchener would have been asked what definition of an unconscious meaning he would lay down. He would not have replied, for he objected in those days to being asked for the 'criteria' of mind, as the phrase then was. Yet certainly there were those heretical ones of us in his Laboratory in 1915 who sought the operational definition of an unconscious meaning and concluded that Titchener was really supporting a behavioral view of meaning.³

5. Since then psychology has come a long way. We now know that operational definitions can always transform a psychological description, expressed in terms of consciousness, into a description in behavioral or physiological terms. One does not have to like operationism to admit this fact. One has only to realize that it can be done whether one likes it or not. Thus everyone is free to choose his set of terms, since the principles for the transformation of one set into the other are known—indeed, have to be known if the terms are to have meaning. It is only for this reason that a text-book, eclectic as to the problem of consciousness, can be written. The Boring-Langfeld-Weld-edited book is such an one. The authors write in terms which are most conventional for the subject-matter in hand, and the tiny sophisticated few, who are worried about the fundamental systematic problems when they read that book, translate automatically. I, for one, always know what an image is in physical operational terms

³ Only at Cornell would this view have been heresy in 1915. At Harvard the behavioral theory of consciousness (meaning) was already explicit: see E. B. Holt, *The Freudian Wish*, 1915.

and I do not care whether the author of a chapter has been bothered by this problem or not. On the other hand, my mentalistic colleague will take the rat's discrimination and know with equal assurance something about his consciousness. There is no difference; we mean the same thing.⁴ And psychology, of course, deals largely with the explanation of meanings, just as Dr. Higginson says, and just as physics deals largely with the explanation of matter.

These five paragraphs are the abstract of my paper on Dr. Higginson's topic. I offer them unexplicated as an antidote to his unwarranted sorrow. Titchener came along nicely, and we have done even better. Pretty soon we shall all seem inadequate, for psychology shall have got beyond even *us*.

[MS. received May 14, 1937]

⁴ Higginson refers (p. 499) to E. G. Boring, H. S. Langfeld, H. P. Weld, *et al*, *Psychology: A Factual Textbook*, 1935, as a book which uses "the approach previously emphasized by Titchener in which mental elements are treated as if they constituted the basic raw materials out of which the whole fabric of mental life is actually woven." That sentence sound marvellously strange to me for this book. Is it justified by the introductory chapter, which has until now been accused only of being too behavioristic, of tacking phenomenal experience on merely as a something extra? Is it justified by the text? No; Higginson says that the text is unable to keep to his formula. Actually I think the noun *sensation* does not appear in the whole text, although the adjective *sensory* is used. We meant to keep *sensation* out, and I at least was amused to think that psychology has now come thus far beyond the Titchener of 1915.

SOME OBJECTIONS TO PROFESSOR CASON'S DEFINITION OF LEARNING

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In a recent article in the *PSYCHOLOGICAL REVIEW*, entitled 'The Concepts of Learning and Memory,' Professor Hulsey Cason has offered the following as a definition of learning: "*Learning is the establishment or strengthening of neural connections between stimulating processes and responding processes as a result of accompanying or immediately preceding psychological acts.*"¹ It is, of course, anyone's privilege to define a field as he sees fit. Definitions of learning, like anything else, would be expected to vary, therefore, in accordance with the idiosyncrasies of the definer. Our own attitude towards the statement of Professor Cason may be due, in fact, to a personal and peculiar failure to interpret his language as he intended. Yet we believe there are others who will read it as we do. In the hope that a free discussion of such matters will make for progress regarding them, we are emboldened to note here a few of the more serious criticisms against Professor Cason's definition.

1. "It is well known that practically all, if not all, learning involves functional connections in the central nervous system . . .," writes Dr. Cason. We might add that it is generally agreed that some sort of structural changes take place in nervous tissue during learning. This, we presume, is what Professor Cason refers to as the "strengthening of neural connections." But whether the changes are in the nature of growth processes, whether synaptic resistance is reduced, whether electrical potentials in different portions of the nervous system are altered, or whether some hitherto unsuspected principle operates, is today almost wholly a matter

¹H. Cason, The concepts of learning and memory, *PSYCHOL. REV.*, 1937, 44, 54-61.

of conjecture. "Practically nothing is known of the structuro-functional changes in nerve tissue which accompany learning," says Freeman of this question.² Is it not over-emphasizing a speculative and theoretical point then, to define learning *as the changes* which take place, when the evidence for such changes is at best indirect and circumstantial? Is it not building a definition about a physiological inference, at the expense of the straightforward and observable facts of behavior?

Learning is first and foremost a change in activity. Yet Professor Cason's statement, as we read it, defines learning solely and exclusively in terms of unknowable nerve processes. It is undoubtedly true that, in the case of higher organisms, neural changes accompany behavior changes and serve as the physiological background for them. From the point of view of psychology, however, it is hard to see how a change in 'neural connections' can be said *to be* the learning.

2. If students of learning accept this definition, and attempt to apply it, they will find themselves in the impossible position of being unable to *prove* that learning can ever take place at all. Stimuli can be presented and responses observed. But no matter how the responses may vary, there will never be any positive evidence of learning unless the 'neural connections' are affected. Will not the only recourse be to annihilate the learner and minutely examine his nervous system, in order to discover whether the 'neural connections,' whatever they may be, have actually been 'strengthened,' whatever that may mean? Since current physiological research has not yet identified the 'connections' it seems there can be little hope of anyone ever demonstrating that an organism can really learn, according to Professor Cason's definition of the term.

Indeed, to speak of learning as the strengthening of connections at all, is reminiscent of the definition advanced by Thorndike when he published his 'Psychology of Learning' 24

²G. L. Freeman, Introduction to physiological psychology, New York: Ronald Press, 1934, p. 488.

years ago.³ But Thorndike did not include in his definition the direct statement that the bonds between situations and responses were neural, although he did give elsewhere his synaptic explanation of the supposedly neurological changes that take place.

3. Comparative psychologists will find still another difficulty with Professor Cason's definition, *viz.*, it eliminates the possibility of learning in lower organisms which do not possess structures that would permit 'neural connections.' Yet simple learning—or something which looks very much like it—has been demonstrated well down the phyletic scale, both in organisms with the rudimentary nerve-net type of transmission system and in some which have no known, genuinely neural, structures for 'connecting' their different parts. A few examples will suffice to illustrate the point.

Certain of the higher plants form what appear to be 'habits' of responding. The common marigold, for example, which opens to light and closes to darkness, can be 'trained' to open and close either in 8-hour or 6-hour rhythms by regulating the illumination accordingly. The new habit, when once established, persists for some time, when the plant is returned to its more normal 12 hours of light and darkness.⁴ Plavilstchikov has recently performed an experiment upon the conditioning of colonies of infusoria, *Carchesii lachmani*, which, to quote Razran, "is the most extensive single experiment in the conditioning of any organism." In the 82 colonies studied, conditioning was established in from 79 to 284 stimulations of light and contact with a glass filament. After this training, parts of a colony could be transplanted to other colonies, without affecting the conditioned response to light of the transplanted parts.⁵ Among the coelenterates and echinoderms, there is also evidence of learning. Thus it has been shown by Fleure and Walton that the sea-anemone, *Actinia*,

³ E. L. Thorndike, *Educational psychology*, Vol. II, 'The Psychology of Learning,' New York: Teachers College, Columbia Univ., 1913, 452 pp.

⁴ C. J. Warden, T. N. Jenkins, and L. H. Warner, *Introduction to comparative psychology*, New York: Ronald Press, 1934, p. 238.

⁵ N. N. Plavilstchikov, *Observations sur l'excitabilité des infusoires*, *Russky Arkhiv Protistologii*, 1928, 7, 1-24. Reviewed by G. H. S. Razran, *Conditioned responses in animals other than dogs*, *Psychol. Bull.*, 1933, 30, 262-263.

whose tentacles can at first be 'tricked' into carrying filter paper soaked in food-juices to the mouth, will learn after several days to reject the paper.⁶ And Preyer⁷ and subsequently Ven⁸ have demonstrated learning in the starfish, as it escapes restraints imposed by the experimenter. Behavior certainly changes in such cases; yet we do not know whether 'neural connections' can be established at all.

Of instances similar to those cited, which could be considerably multiplied, Maier and Schneirla have recently written, "They are primitive forms of 'learning,' since they are the product of the animal's experience as it temporarily alters the irritable and contractile tissues, and since the nature of the behavior is altered during this interval."⁹ Miss Washburn may also be quoted in this connection. She writes, "It is sufficiently clear that animals have the power of learning, in the sense of the capacity to react differently to a present stimulus because of their past experience with it. Probably not a single animal form is so low that it lacks this power."¹⁰ While it is true that there is additional and controversial evidence, not cited here, to the effect that lower forms may not be able to learn, we cannot scientifically ignore well-established observations like those mentioned. Unless Professor Cason's definition is meant to apply only to man, and to man's biological near-relatives, it should therefore be broadened to include the possibility of learning in lower organisms.

To our way of thinking, it would be more exact to keep the definition of learning on a purely objective and descriptive level. If neurological implications are omitted then there would be no danger of going beyond the neurological conditions, whatever they may be. Controversial or unsettled

⁶ H. J. Fleure and C. L. Walton, Notes on the habits of some sea-anemones, *Zool. Anz.*, 1907, Bd. 31, S. 212.

⁷ W. Preyer, Ueber die Bewegungen der Seesterne, *Mitth. a. d. zool. Stat. zu Neapel*, 1886, Bd. 7, S. 27, 191.

⁸ C. D. Ven, Sur la formation d'habitudes chez les astéries, *Arch. néer. physiol.*, 1922, 6, 163.

⁹ N. R. F. Maier and T. C. Schneirla, Principles of animal psychology, New York: McGraw-Hill, 1935, p. 54.

¹⁰ M. F. Washburn, The animal mind, 4th ed., New York: Macmillan, 1936, p. 328.

issues should, of course, be discussed when the topic is gone into in detail. But they do not belong in a generalized introductory statement. The simple descriptive facts can be stated without ambiguity or speculation. In a single word, learning is *modification*. In three words, it is *modification of response*. In a few more words, it is *a persisting change or modification of behavior which results from repeated or continuous stimulation*. The term 'persisting' in this definition eliminates any possible confusion between learning, conditioning or habit on the one hand, and sensory adaptation or fatigue on the other hand. Although modifications are produced by fatigue and sensory adaptation they are of short duration when compared to the changes resulting from learning. 'Repeated or continuous stimulation' is necessary in every case of learning—including those cases in which something is learned 'upon a single trial.' For even if learning can actually occur in one immediate presentation of a stimulus, it can never be shown to have occurred unless the stimulation is repeated (or continued long enough) to demonstrate that the change has taken place.

To sum up then, we find Professor Cason's definition of learning unacceptable for the following reasons:

1. It overemphasizes a physiological inference at the expense of more fundamental and directly observable facts of behavior.
2. It places the evidence of learning within the body itself. As a result, learning can never be proven to have occurred without examining the nerve tissues.
3. It denies the possibility of learning to organisms without a nervous system similar to that of man—a denial which at the present time is not supported by sufficient evidence to be accepted as fact.

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DR. KELLOGG ON THE DEFINITION OF LEARNING

BY HULSEY CASON

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I would like to express my thanks to Dr. Kellogg for the favor of his free and hearty discussion of my definition of learning. Dr. Kellogg and I are in complete agreement that the term learning should be defined, and that the particular words used in the definition are important, and Dr. Kellogg has done much better than those who flippantly assume that a definition of learning is not necessary. Dr. Kellogg's paper and my note will end the discussion between us in the present journal, and this is perhaps as it may well be, because I think that after Dr. Kellogg reads the present note he and I will be in substantial agreement on all essential points.

Psychological definitions.—Dr. Kellogg remarks that "It would be more exact to keep the definition of learning on a purely objective and descriptive level," but the term 'objective' has a dualistic reference, and there are some psychological descriptions which are not 'objective.' It is also fairly common for definitions to include both explanation and description.

On theoretical grounds and in the interests of future research in scientific psychology, it seems desirable to remain or move as close as possible to our best scientific friends, physiology, neurology, endocrinology, and anatomy, because it is only in this way that we can make proper use of what is known about the organic conditions and processes of the body. All psychologists refer to parts of the body when they are describing sensations and reflexes, and everyone assumes that psychological processes occur within the skin. The principal thesis of what I have called Organic Psychology¹ is that one should take account of what is known about the psychological

¹ H. Cason, The organic nature of sensations, *J. Gen. Psychol.*, 1937, 16, 357-377.

and other related organic processes in the body; and the question may be raised whether any psychological process is ever well understood and explained when—because of faith in one of the psychological schools which come and go—one limits one's self to a description of explicit behavior.

My organic definition of learning.—I have tried to relate my definition of learning to the psychological processes which occur in the body, and have referred to stimulating processes, neural connections, and responding processes. If one is studying a particular form of learning in a human or sub-human subject, one should mention the form of learning, the kind of subject, where the stimuli are applied, and what kind of response is observed or measured. In other words, in studying a psychological process, one should whenever possible and practical study the psychological process itself.

In the diagnosis of an ordinary medical disease, some use may be made of the verbal reports of the subject, the readings of a thermometer, and the analyses of blood, urine, and so on. That is to say, various superficial symptoms may be used in the diagnosis of a disease, but the disease itself and the symptoms of the disease are very different things. In the same way and for similar reasons, the learning process itself and any explicit behavior which may be involved in the learning are also different matters.

The evidence for learning.—Learning is ordinarily demonstrated by a reproduction test in somewhat the same way that a disease is ordinarily diagnosed by observing symptoms; but just as the symptoms of the disease are not the disease itself, so any explicit behavior which may be present during learning, or during reproduction, is not the learning process itself. A practising physician may diagnose measles without studying and without knowing much about the physiological processes inside the patient, and the fact that learning has occurred is also generally demonstrated in the complete absence of any kind of physiological research.

When Dr. Kellogg suggests that my definition of learning "places the evidence of learning within the body itself," he neglects the important distinction between the learning pro-

cess itself and the signs or indicators which are ordinarily used in demonstrating that learning has occurred. The psychological processes that are present in the subject's body during a reproduction test are different from the psychological processes that were originally present in him when the learning occurred.

Learning in the lower animals.—I have never thought and have nowhere said that learning occurs only in human subjects, and my definition of learning is purposely designed to include the learning processes of normal and abnormal adults and children and practically all subhuman animals. My definition says nothing, however, about the various kinds of neural tissue, and it is equally silent on what parts of the nervous system are most involved in learning. I do not think there is any disagreement here, because Dr. Kellogg and I are both aware (1) that most lower animals, especially those of any psychological importance, have neural functions, (2) that some animals fairly low in the phylogenetic scale can learn a little, although very little, and (3) that neural functions always seem to be involved in the kind of processes regarded by practically everyone as learning processes.

The usefulness and appropriateness of all psychological definitions fade at the lower end of the phylogenetic scale, and perhaps the reason for this is that when one descends to such low levels, he has left the principal field of psychology and has become interested in the concepts and vocabulary of the admittedly fascinating and important, but still largely non-psychological, fields of invertebrate zoology, protozoology, bacteriology, and botany.

Neural connections.—It has long been known that neural functions are involved in sensations, reflexes, and other bodily processes, and I have taken the liberty of assuming that the scientific evidence justifies the conclusion that the essential processes in learning are neural in nature, although I realize that other organic-psychological processes are also involved.² Perhaps the majority of the disciples of behaviorism would prefer no reference to neural functions on any occasion, and

² H. Cason, *Organic psychology II: The psychological organism*, *Psychol. Rev.*, 1934, 41, 356-367.

in my definition of learning one could substitute the word 'functional' for the word 'neural' if one so desired; but it is my opinion that referring to neural functions in a definition of learning is not just a matter of taste. One may or may not mention the heart in a definition of circulation, or any endocrine functions in descriptions of menstruation and pregnancy, or the nervous processes involved in sensations and reflexes in explanations of the sensations and reflexes,—but it would seem that in a definition of a psychological process, the only result of referring to other known-to-be-involved-and-closely-related organic processes in the body (in this case, neural processes) would be an increase in clarity and understanding. By referring to neural processes we do not assume that everything is known about them, although a good deal is known, and much of what is known is important. If it were permissible to speak only of matters about which everything were known, one's speech would be limited indeed.

Organic states and conditions included in Dr. Kellogg's behavioristic definition of learning.—The difficulty with a severely behavioristic version of the learning process is apparent in Dr. Kellogg's proposed alternative definition, which suggests that learning is 'a persisting change or modification of behavior which results from repeated or continuous stimulation.' I think Dr. Kellogg will agree that in the parthenogenetic reproduction of an ovum cell in a salt solution, there is "a persisting modification of behavior" which results from "continuous stimulation." But in this case there is no forgetting! And I believe that Dr. Kellogg, after further consideration, will also agree that his proposed definition of learning includes (1) the growth processes of animals and plants that are influenced by "repeated stimulation," (2) the permanent injuries of sense organs (*e.g.*, hearing) from intense and "repeated stimulation," and (3) the permanent injuries of certain organs of the body (*e.g.*, the heart) from prolonged overwork. And do we not also have "a persisting change or modification of behavior which results from repeated or continuous stimulation" in the case of each of the following: (A) excessive localized muscular fatigue from repeated exer-

cise, (B) general fatigued, exhausted, and neurasthenic states from prolonged overwork, (C) the tonic condition of muscles, (E) prolonged hunger and thirst, (E) continued overeating, (F) the iodine salt treatment of hypothyroidism, and (G) a person dying from the long continued use of large quantities of alcohol? Each of these cases involves a special organic state which may continue longer than some learned material is retained. Dr. Kellogg's proposed definition of learning includes a number of organic states and conditions which are not ordinarily regarded as illustrations of learning.

Common forms of learning not included in Dr. Kellogg's definition.—Since Dr. Kellogg's definition of learning includes the words "which results from repeated or continuous stimulation," his concept also fails to include several forms of learning which I believe practically everyone regards as forms of learning. I have in mind those cases of learning where the associations are formed in one or in only a very few repetitions of the pattern stimuli. The formation of associations may be quite rapid in the learning of paired associates, conditioned emotional responses, and in ordinary observation, reading, and listening to another person speak. It has even been suggested by some students of learning that when the conditions are favorable, practically all learning is the result of one or at most relatively few repetitions of the pattern stimuli.

In general it seems more desirable at least to attempt to define learning in such a way that it will include the widely recognized forms of learning in the higher animals than to strain the concept to such an extent that it will include the alleged modifications of 'behavior' in the marigold plant, infusoria, coelenterates, and what not. Some nice psychological results may some day come from these studies of the lower forms of life, but nicer results are already coming, and coming faster, and coming in greater quantity, from the studies of learning in higher animals and especially from the studies of verbal and other symbolic forms of learning in human subjects.

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